THE VETERINARY RECORD

FOUNDED BY WILLIAM HUNTING, F.R.C.V.S., 1888

No. 46. NOVEMBER 15, 1952 VOL. 64.

PAPERS PRESENTED TO CONGRESS, B.V.A., 1952

No. 3.—FELINE ENTERITIS*

BY

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Miss M. Dalby, B.V.SC., M.R.C.V.S., of Menston-in-Wharfedale, near Leeds, presided over a good attendance at the session in the Royal Hall, Harrogate, on the morning of the Wednesday of Congress week, devoted to the consideration of Dr. Gledhill's paper on "Feline Enteritis." The Recording Secretary was Miss C. A. Woods, M.R.C.V.S., of Menston-in-Wharfedale.

The CHAIRMAN, in opening the meeting, called upon Dr. Gledhill to introduce his paper on feline enteritis. Cats were a problem to them all, however unwilling they might be to accept it, so that this paper would be a great help.

Dr. GLEDHILL: In the few moments at my disposal I wish to refer particularly to the virus diseases of cats which have been confused with feline enteritis, rather than add to anything I have said with regard to feline enteritis, but perhaps you would forgive me if I first refer briefly to the nature of the viruses in general and their characterisation because it bears very much on the problems before us, particularly the problem of treating cats. I am thinking particularly of feline enteritis and the respiratory diseases of cats, all of which are caused by viruses.

Viruses are defined by the two characters of being small and filter-passing on the one hand, and by their incapacity to grow on artificial media in the absence of living cells on the other hand. There are organisms which possess only one of these characters. Thus, the pleuro-pneumonia organisms, although filterable, are capable of growth in culture media. These are not regarded as viruses to-day. There is the rickettsia group of which some are not filterable yet they require living cells for growth. This group is not classified among the viruses. The viruses themselves possess both characters, namely, they are filterable and they grow only in living cells. They range in size from 300 mμ down to 10 mμ in the case of the foot-and-mouth virus. The larger viruses, belonging to the psittacosis-lymphogranuloma group and just visible by the ordinary light microscope, are susceptible to several of the antibiotics, in particular aureomycin, terramycin, chloromycetin and penicillin. In contrast, no antibiotic yet discovered has proved to have a specific action upon the smaller viruses. However, improvement in the clinical condition can arise from treating any secondary bacterial invaders with antibiotics and thereby often be of practical value in treating the natural disease.

Feline enteritis is caused by a small or middle-sized virus, 80 to 100 mμ in size, and one would not expect antibiotics to have any action upon it. On the other hand, cat influenza—if one may use that name for the syndrome of rhinitis and coryza—appears to be caused by two viruses producing diseases which are distinguishable. Both are of much larger size and are likely to be capable of cure by antibiotics.

I would like to give you a little more information with regard to these two respiratory diseases which we have found to be due to different agents. In the first place there is the type of disease in cats in which there are symptoms of rhinitis and coryza with conjunctivitis with no febrile reaction. The cat is rather depressed and recovery is somewhat slow; the mortality is practically nil. On *post-mortem* examination there is usually extensive pneumonia and inflammation of the turbinates. It is possible to obtain an extract from the turbinates and lungs with which one can infect mice. Elementary bodies are present which may be seen after staining suitable preparations. The agent in mice is susceptible to penicillin but not to the sulpha drugs and in all these respects it resembles the disease described in the United States by Baker in 1944 and which he called feline pneumonitis.

In the other respiratory disease likewise the most prominent symptoms are severe rhinitis and coryza. The condition is more acute and there is a temperature response ranging from 104° to 106°. Under field conditions some affected cats may die but under the conditions in which we have kept the cats the mortality is low. That it is a separate disease and not merely a more acute manifestation of the former disease appears to follow from the following facts: first, it is impossible to isolate the agent in mice; secondly, it is impossible to grow the agent in fertile eggs, at least we have been unable to do so; thirdly, we have never seen elementary bodies in smears and sections of turbinates of affected cats; fourthly, pneumonia is not an obvious lesion, being absent or very limited in extent; fifthly, there seems to be no cross immunity between the two diseases, although it is not easy to reach a conclusive result since in both only a partial immunity follows upon attacks.

I think many cats carry these viruses for a long time. Occasionally in feline enteritis one encounters acute rhinitis and coryza. I believe that these symptoms may result from relapses in cats affected with feline enteritis which were carrying one or the other respiratory virus. The appearance of such symptoms with feline enteritis may have given rise to a certain confusion and a tendency to regard them as a normal manifestation of feline enteritis. There are two clearly marked conditions, feline enteritis on the one hand and the respiratory diseases on the other.

With regard to feline enteritis, it has come to my notice that there is now apparently a vaccine obtainable in Britain against it and from the experimental work which has been done the possibility of making such a vaccine has long been recognised. The difficulty has no doubt been a practical one, as I have mentioned in my paper.

Dr. Gledhill's paper was as follows:—

There is widespread confusion in regard to the infectious diseases of cats, due mainly to insufficient study of their pathology. The researches of Hindle and Findlay (1932) upon feline enteritis (called distemper by them) and of Dalling (1935) established a filtrable virus as the aetiological agent. Hindle and Findlay (1932, 1933) tended to regard the virus as the primary cause of other clinical syndromes, while

* Presented to the 70th Annual Meeting of the British Veterinary Association, at Harrogate, on September 10th, 1952.

Dalling thought, on the basis of cross immunity tests, that the common respiratory syndrome of cats, now generally called feline influenza, was due to a different agent. At Mill Hill, Drs. Andrewes, Niven and I have done some work on this respiratory syndrome and have obtained evidence of two diseases due to distinct viruses both of which appear to differ from the aetiological agent of feline enteritis. Both these forms of feline respiratory infection are characterised by rhinitis and conjunctivitis, and by their rather long course (21 to 28 days), leading generally to recovery; feline enteritis, on the other hand, is characterised by absence of coryza and lacrimation, by its shorter course and fatal termination. Since Dalling's publication (1935) there is little evidence of further progress by British investigators. In the United States, however, Lawrence and Syverton (1938) and, independently, Hammon and Enders (1939a) have described a disease pathologically characterised by severe disturbance of hematopoiesis and by the presence of eosinophilic intranuclear inclusion bodies in the epithelial cells of the intestine and elsewhere. Severely ill cats show such marked leucopenia and relative lymphocytosis that accurate diagnosis may be based upon blood counts. The disease has been given several names, all of which refer to the blood picture, namely, infectious feline leucopenia, malignant panleucopenia and infectious feline agranulocytosis. The outward symptoms of this disease, which I shall call infectious leucopenia, do not differ from those of feline enteritis and there are other points of resemblance which will appear later. As American and continental workers have gained a considerable knowledge of infectious leucopenia, it becomes a matter of urgency to determine whether the feline enteritis of Britain is really infectious leucopenia. In the course of this communication I hope to present you with the similarities and differences. I, personally, think that the diseases are probably the same but that there is insufficient knowledge of feline enteritis to accept their identity without some reservation.

Symptoms and Pathology of Feline Enteritis

Symptoms.—The sudden onset of the disease is characterised by profound depression and dehydration. The temperature is somewhat elevated early in the disease (103° to 105°) but severely ill cats generally show a subnormal temperature. Vomiting of small quantities of bile-tinged mucus often occurs and more rarely diarrhoea develops. Coryza and lacrimation are very seldom observed. It is possible that in the few cases of feline enteritis showing coryza and lacrimation, these symptoms are due to simultaneous infection with the agent of a respiratory infection of cats, arising either extrinsically or by the cat itself suffering a relapse towards an earlier respiratory infection, since cats recovering from these infections do suffer relapses. Death may occur rather unexpectedly within two or three days. Sometimes cats are found dead without premonitory symptoms having been observed and, in other cases, cats may survive longer than three days. It is possible that the virulence of strains differs considerably and thus gives rise to variable survival times, although all strains eventually seem to kill the majority of cats showing symptoms. For example, Hindle and Findlay's strain "Siamese V" gave a survival time of nine days (10 cats) as against 21½ days for strain "Siamese IX" (six cats).* The mortality in cats developing symptoms is 80 to 90 per cent.

* These averages were calculated from Hindle and Findlay's data (1932, *op. cit.*, p. 17 and p. 19), and examination of them shows the averages to differ significantly ($t = 3{\cdot}9$, $n = 14$, $p < 0{\cdot}01$).

Pathological Changes.—Congestion of some portion of the small intestine is usually seen both on the serosal and mucosal surface, most frequently in the middle region of the small intestine. Microscopic examination shows congestion of the mucosa in early cases. Later cases may present a picture of haemorrhage, sloughing and destruction of the mucous membrane. In extreme cases, the mucosa is covered by a fibrinous deposit which contains erythrocytes, desquamated epithelium and bacteria. Infiltration with neutrophil leucocytes is not a prominent feature. Hammon and Enders (1939a) describe for leucopenia a somewhat similar pathology of the intestine with the exception that they note the presence in the epithelial cells of rather large intranuclear eosinophilic inclusion bodies. They do, however, state that these inclusions cannot be seen if the autopsy is carried out several hours *post mortem*.

The mesenteric lymph nodes are swollen and moist. According to Hindle and Findlay (1932) they often show haemorrhages. They also note that these haemorrhages are sometimes absent but in such cases the sinusoids contain large numbers of erythrocytes and neutrophils in various stages of degeneration. For infectious leucopenia, Hammon and Enders (1939a) note an extensive oedema and widening of the sinusoids together with a marked reduction in the number of lymphocytes and injury to the cells forming the germinal centres. Intranuclear inclusion bodies like those found in the gut are also found in some of the cells of the germinal layer. They also mention that the sinusoids are crowded with mononuclear phagocytes and erythrocytes.

Congestion of the lungs is sometimes observed. Microscopically, such cases reveal congestion of the alveolar capillaries which may rupture and fill the alveoli. Some alveoli and bronchi contain a fibrinous exudate with a few mononuclear cells. Neutrophils are absent and the bronchial epithelium remains intact.

Enlargement of the spleen, pleurisy with effusion, peritonitis and a haemorrhagic thymus were other pathological changes noticed by Hindle and Findlay.

Hindle and Findlay (1932) make no mention of any abnormality of the bone marrow. They did, however, make blood counts in a certain number of cats; their earlier results suggesting a monocytosis were not confirmed by subsequent counts. Jennings (1947) noted that in place of the rather solid column of red bone marrow in the long bones of kittens, he found a semi-liquid mass. Microscopic examination showed the tissue to be aplastic. The few cells present were mainly erythrocytes and normoblasts. Although a few myeloblasts were seen, mature granular leucocytes were absent. Moreover, Jennings found leucopenia, usually severe, in all cats seen by him prior to death—these amounted to 15 natural and two experimental cases. Generally, there was also a relative lymphocytosis. This picture closely resembles that described for infectious leucopenia by the United States workers.

Can Feline Enteritis be Identified with Infectious Leucopenia?

Feline enteritis as described by Hindle and Findlay bears a close resemblance to infectious leucopenia as described by the American workers in symptoms and even in pathology, apart from the noticeable absence of an aplastic bone marrow, leucopenia and eosinophilic inclusion bodies. Hindle and Findlay made blood counts in some of their cats and looked for but failed to find inclusion bodies. Fifteen years later Jennings described a disease, presumably from cats in and around Liverpool, which corresponded to infectious leucopenia in respect of the aplastic state of the bone marrow

and the consequent leucopenia. Moreover, he found eosinophilic intranuclear inclusion bodies in some cases. Finally, in cases of feline enteritis which have occurred among cats kept at the National Institute for Medical Research, we have noted the aplastic state of the bone marrow and have found intranuclear inclusion bodies (Andrewes, Gledhill & Niven, unpublished). Thus, it is reasonable to suppose that feline enteritis, at the present time at least, is in fact usually infectious leucopenia. This view is rendered more probable by the fact that infectious leucopenia has been found to occur in Germany and France (Kikuth, Gönnert & Schweichert, 1940 ; Levaditi, Lépine & Verge, 1948). Confirmation should be sought by comparing an authentic United States strain of the virus with British strains. Whether the disease of Hindle and Findlay may be identified with infectious leucopenia is more doubtful. In spite of their not observing leucopenia nor intranuclear inclusion bodies, I think, nevertheless, that their disease may have been infectious leucopenia. In France, Pavilanis (1948) has noted both primary leucocytosis and leucocytosis following upon leucopenia (probably as a result of secondary bacterial infection) in 16 per cent. of cats infected with infectious leucopenia. Under such conditions, blood counts of a comparatively few infected cats might easily lead to confusing results. The same author (1947), in stating that a strain from Lyons is less virulent than his Parisian strain, mentions that in the former inclusions are less frequent. The survival times of Hindle and Findlay's cats are longer than usual in two of their three strains, so that the low virulence might have made the inclusion bodies too infrequent to be observed. Added to this is the fact, already mentioned, that the inclusions are not observed if the autopsy is delayed *post mortem*. It is not without interest that Lawrence, Syverton and others (1943) isolated infectious leucopenia virus from the tissues of two cats which had been ill of a disease diagnosed as feline enteritis by " two competent diagnosticians " from leading United States veterinary schools.

Properties of the Infective Agent.—That the primary causative agent of feline enteritis is a filtrable virus was first indicated by Verge and Cristoforoni (1928) and amply confirmed by Hindle and Findlay (1932). The latter authors showed that suspensions of the virus in 50 per cent. glycerol retained virulence while extracts dried over P_2O_5 at laboratory temperature lost their capacity to produce overt disease but, nevertheless, seemed able to immunise cats. Syverton, Lawrence and others (1943) found infective leucopenia virus in 50 per cent. glycerol buffered at *p*H 7·2 still viable after 138 days and freeze-dried virus stored at refrigerator temperature retained its full virulence.

The size of the leucopenia agent has been estimated by ultrafiltration through gradocol membranes and found to be 80 to 100mμ (Lépine & Pavilanis, 1948).

Mode of Spread.—Cats may be infected by practically all routes of inoculation. Thus, Hindle and Findlay (1932) infected cats by intranasal spraying and intracerebral, subcutaneous and intraperitoneal injection. Syverton, Lawrence and others (1943) confirmed these results (except the intracerebral route, which they did not undertake) and, in careful experiments, showed infection to occur by ingestion of infected material. They also showed the virus to be present in the blood, most tissues, nasal secretions and faeces. It was also sometimes present in the urine. What is of particular interest is that the virus may be present in the blood during the incubation period, 48 hours after infection, as well as after cats have become ill. Presumably, therefore, the disease may be spread at cat shows by cats which appear quite normal.

A very characteristic feature of the disease is its extreme infectiousness. Hindle and Findlay (1932) showed that cats readily contract the disease by contact and by fomites. Hammon and Enders (1939a) note that " healthy animals brought to the laboratory and observed for a period of time varying from 8 to 18 days, either spontaneously developed characteristic symptoms or later proved refractory to the inoculation of material from sick animals or to contact exposure to cats suffering from the infection." A similar observation is explicitly noted by Pavilanis (1949) and, indeed, it has been my own experience in cats held at the National Institute for Medical Research. Cats isolated in a manner which has proved sufficient to prevent the spread of canine distemper in dogs and ferrets, failed to prevent cases diagnosed as feline enteritis. Several cats (out of about 30) widely separated from each other developed symptoms and nearly all the remainder resisted subsequent challenge with the agent from earlier cases. To explain findings of this kind, Hammon and Enders (1939a) suggested the possibility of an arthropod vector and Syverton, Lawrence and others (1943), thinking along this line, showed that the conditions for arthropod transfer of virus from cat to cat are fulfilled : the virus is present in the blood of infected cats during the periods of incubation and illness, as already stated, and very small doses of virus introduced into the skin cause the disease. So far as I am aware, there are no published results of attempts to follow up this possibility. It is, perhaps, suggestive that Smith (1949), in a survey of 574 cases around Boston, Mass., found the disease to be enzootic throughout the year, except in July, August and part of September, when it assumed epizootic proportions. At Mill Hill we have pursued the idea that virus might be carried in Ascaris eggs ; but the few experiments we did yielded no conclusive result.

Species Susceptibility.—Feline enteritis has never been observed in species other than cats, or at any rate domestic cats and wild species of the Felidae. In regard to the latter, Hindle and Findlay (1932) refer to deaths of imported animals at London Zoological Gardens in which the symptoms and lesions suggested feline enteritis but, so far as I am aware, no attempt has been made to transfer virus from these sources to domestic cats. These workers attempted to infect the ferret, mongoose, rabbit, guinea-pig and mouse by intraperitoneal inoculation without producing symptoms or lesions in them. Nor were mice susceptible to intracerebral inoculation.

Working with the virus of infectious leucopenia, Hammon and Enders (1939a) showed the rabbit, guinea-pig, ferret and mouse to be insusceptible and Kikuth, Gönnert and Schweichert (1940) obtained negative results with the dog, rhesus monkey, rabbit, hamster, canary, hedgehog and rat. In view of the small number of animals employed and the method of " blind " serial passing not having been used, Syverton, Lawrence and others (1943) attempted to adapt the virus to rabbits, mice, guinea-pigs and ground squirrels by using four to six blind passes, employing intracerebral, intranasal and intraperitoneal routes of injection for suspensions of brain, liver and spleen and intestinal washings for each species. No symptoms or lesions were produced in any of these species nor were cats infected by inoculation of tissue extracts from the animals employed for the last passage transfer.

Natural and Artificial Immunisation of Cats

Practically all who have worked with feline enteritis or infectious leucopenia testify to the fact that many cats are insusceptible to infection—about 30 per cent. in an enzootic area (Pavilanis, 1949). In attempting to avoid this difficulty the tendency has been to purchase cats as young as possible

from private homes, so that most of the work has been done with animals of less than six months of age. An appreciable proportion of such cats are insusceptible and the proportion rises the longer that they are kept in the animal house before being used. Dalling (1935) states that cats, however young they may be, are frequently insusceptible when obtained through ordinary sources. The suggested explanation of this immunity without previous signs of illness having been shown is that sufficient maternal antibody is acquired *in utero* and early lactation to enable kittens to limit natural infections to inapparent immunising infections (Enders & Hammon, 1940). If this be so, one cannot help wondering whether immune mothers may not be carriers, infecting their young soon after birth or even *in utero*. Such ways of infection are believed to occur in several virus diseases of mice—for example, lymphocytic choriomeningitis. In the case of a mouse hepatitis described by us recently (Gledhill & Andrewes, 1951) about 70 per cent. of an infected colony of mice are actively immunised by weaning time (three weeks after birth) as a result of infection acquired from their mothers soon after birth and possibly *in utero* in some cases. If such a mechanism does operate in the case of feline enteritis, it might offer the possibility of immunising entire litters by immunising mothers with killed vaccine before pregnancy, followed by virus during pregnancy. But as a preliminary to investigating this possibility, it would be necessary to establish that recovered animals do commonly harbour the virus. It would seem that this is the case, to account for immunity developing in cats held in contact with other cats. It is not without interest that Dr. Niven has observed intranuclear inclusion bodies, resembling those of feline enteritis, in cells of the nasal epithelium of normal cats.

Another possible explanation of the high proportion of immune cats which have had no history of previous illness, is that contact infection with avirulent immunising virus may take place. This hypothesis may appear too wild for serious consideration. Nevertheless, it would explain the rise in the number of immune cats in proportion to the period of time that they are kept and would especially account for those cats too old to have retained maternal antibody becoming immune without showing symptoms. Moreover, Hindle and Findlay (1932), describing the induction of immunity by dried virus, ascribe the effect to avirulent virus rather than inactivated virus.

These intriguing possibilities might be fairly simple to investigate if mice or fertile eggs were susceptible to the virus of feline enteritis. It would seem feasible to attempt their investigation if a virus-free strain of cats could be bred for experimental purposes, as was done for canine distemper with dogs by Laidlaw and Dunkin. However, it does not yet seem to have proved possible to breed cats satisfactorily in complete captivity. Without such cats the best planned experiments would be likely to yield only inconclusive results (cf., Enders & Hammon, 1940).

Dalling (1935) claimed that cats may be immunised by virus inactivated with formalin. He suggested that vaccination of cats should be followed by their inoculation with virus to give them a more durable immunity. Enders and Hammon (1940) and Brion and Bertrand (1948) have brought evidence indicating that formalised virus can confer an active immunity. As regards passive immunity, Hindle and Findlay (1932) showed that relatively large doses of serum from recovered cats protected most of the test cats against challenge with virus. Enders and Hammon (1940) obtained a similar result with their virus, and Lawrence, Syverton and others (1943) successfully protected cats against challenge with their virus by passively immunising them with serum from convalescent cats submitted to them by Enders and Hammon. Pavilanis (1949) showed the virus to be neutralised *in vitro* by the sera from a "normal" and from an immune cat but not by the serum from a cat dying of the disease.

These results would suggest that it would be possible to make a commercial product for immunising cats and you may wonder that nothing is yet available in Britain. I have no doubt that the answer to this is that producers are discouraged by their inability to secure uniformly susceptible cats for making and testing their products. Although vaccine followed by virus would probably be the more reliable method, a method depending upon the inoculation of serum and virus might be economically more feasible since it would enable the cats found to be insusceptible to the virus to be used for serum production. The production of immune serum for giving protection over short periods of time, such as for cat shows, would seem to present only the difficulty of testing it.

Is Feline Enteritis a Fitting Name for the Disease?

There has been a difference of opinion between Hammon and Enders (1939a) on the one hand and Lawrence, Syverton and others (1943) on the other hand as to the name for what I have called infectious feline leucopenia. The latter authors, after giving their reasons for preferring the name "infectious feline agranulocytosis" to "malignant panleucopenia," go on to say that the name "feline enteritis" is not so readily disposed of since it represents a clinical entity accepted by veterinarians. They do suggest disposing of it, nevertheless, on the ground that it may be a clinical designation for a variety of maladies. Recognising the latter possibility, I would argue that since one of such variety of maladies has now become pathologically and aetiologically defined, this defined malady should take the name of feline enteritis exclusively, giving some unappropriated name, such as distemper, for the other clinical syndromes which simulate it. I should have thought that this is the usual procedure, followed, for example, in the case of human influenza. Hammon and Enders (1939) object to "feline enteritis" in that the gut lesions described by them cannot truly be regarded as an enteritis. While this may be true, I do not think it is sufficient reason for displacing a name which is already in currency —though I have the impression that "feline enteritis" is being displaced by "panleucopenia" in the States. Furthermore, since Pavilanis (1948) claims that 16 per cent. of infected cats show a leucocytosis, perhaps "panleucopenia" and "agranulocytosis" are not, after all, perfect names. At any rate, the question does not arise for us until we have finally established that the agent causing feline enteritis in Britain is closely related to that which causes infectious leucopenia in the United States.

Summary

After distinguishing feline enteritis from the predominantly respiratory diseases of cats, an attempt is made to review the epizootiology, symptomatology and pathology of feline enteritis of Great Britain in relation to infectious feline leucopenia, first described and accurately characterised by workers in the United States. On the basis of the facts, the opinion is expressed that the clinical syndrome termed feline enteritis now occurring in Britain is probably the same as feline leucopenia of the U.S.A. Whether the disease described by Hindle and Findlay in 1932 is also feline leucopenia is rather more doubtful but, nevertheless, the author inclines towards the view that it is.

The properties of the aetiological agent of feline enteritis are described. Its high infectivity and the obscurity in its

mode of spread are stressed. Many young cats are immune to feline enteritis and, of these, very few indeed have any history of previous illness. This immunity is thought to result from chance infections at a sufficiently early age for kittens to be protected from overt disease by antibody received from their mothers. The author suggests as a possibility that immune mothers may transfer virus as well as antibody to their offspring, *in utero* or during lactation. An alternative explanation of the high incidence of insusceptible cats would be that naturally transferred virus may often be avirulent yet capable of immunising and this explanation is better able to account for older cats developing immunity without showing symptoms. Investigation of these hypotheses, handicapped by the insusceptibility of laboratory animals to the virus, is rendered almost impossible by the insusceptibility of so many cats. Immunisation by formalised virus followed by active virus has yielded promising results but their preparation on a practical scale is rendered difficult by the problem of obtaining uniformly susceptible cats for testing them.

The suitability of the name "feline enteritis" for the disease is discussed. It is considered that it should be retained even if feline enteritis had been identified with infectious feline leucopenia; but the question does not arise in Britain as long as there is some doubt as to whether the diseases are really the same.

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DISCUSSION

The discussion was opened, as follows, by Miss W. M. Brancker, M.R.C.V.S., of Sutton Coldfield:—

I wish to thank the Provisional Committee for the honour of being invited to open this discussion. The invitation rather puzzled me: then I realised that the Committee were expecting a very learned paper from Dr. Gledhill and they wanted an opener who could be guaranteed to bring the paper down to earth. Their assumption was correct and Dr. Gledhill is to be congratulated on producing a highly scientific and yet extremely readable paper on a subject which has previously been regarded by most members of the profession and of the public, as of little significance. He is also to be congratulated on putting the results of his work into comparatively simple language. I am not in a position to comment on the pathological details in the paper: I hope the research workers present will do this, and I feel it would really be more useful if these opening remarks were complementary to Dr. Gledhill's paper. I shall, of course, criticise and question where the clinical symptoms and the practical application of the laboratory work is under discussion.

The majority of people probably regard this subject as of interest only to those in small-animal practice. Dr. Gledhill, I suspect, regards feline enteritis as another virus disease to be conquered in the interests of mankind and science, but it has an even wider significance. Cats are of considerable economic importance as vermin killers on farms, in granaries and among storage dumps and feline enteritis is a highly infectious and lethal disease of cats which is capable, if a widespread epidemic occurred, of greatly reducing the number available for such work and so endangering the food stocks of the country; yet it is extremely difficult to control this disease because the susceptible animals are so difficult to control themselves. Cats are classed among the domestic animals but personally I always feel that this classification should be in inverted commas. An enormous number of cats owe no allegiance to any particular place or person and only take from man what can be obtained without obligation. Even the household pet is usually an independent creature and the majority are clever enough to appear stupid. Cats not only wander over the British Isles but despite the quarantine regulations which control the more aristocratic members of this family there must be many sea-going cats who take unofficial shore leave. As a clinician I should have thought that the American disease referred to by Dr. Gledhill as infectious leucopenia was of common stock with the disease known as feline enteritis in England, but I quite appreciate that it is necessary to prove these points and not to jump to conclusions.

Symptoms.—In a typical case seen early there is almost invariably a temperature; on examination there is evidence of abdominal pain and the cat usually crouches without "turning in" the front paws. Frequently the cat crouches over a bowl as though thirsty but unable to lap. Nasal and/or ocular discharge may be present and possibly some salivation but these symptoms usually only occur in a cat which has been "off colour" for at least 24 hours. There is generally stasis of the bowel. Temperature taking in a cat may be misleading as they are extremely susceptible to external heat and during the day in summer may easily run a temperature of 103 or 104. Excitement will also send up the temperature and the thermometer readings of those cats with an addiction to the hot water tank or the kitchen stove should be taken with some reservation.

Those cats which survive the first 48 hours may develop secondary infections but in uncomplicated cases there is little change in the symptoms. On or after the third day diarrhoea usually occurs and this will persist for several days even in cases that do not end fatally. The motions are loose but not very frequent and on the fifth or sixth day there is often evidence of haemorrhage. Occasional vomiting will occur for 7 to 10 days. Some cats will lap throughout the whole course of the disease while others will not attempt to drink until they are definitely beginning to improve. This may depend on whether the cat normally drinks water or not—those animals which usually drink milk will not have any desire for such a cloying drink and yet will not turn instinctively to water. The return of the appetite again appears to be largely a matter of temperament.

A week after the onset of symptoms the mucous membranes begin to look pale and this condition is usually progressive for about a week, by which time the membranes are practically colourless and cats which survive the acute stage of feline enteritis will frequently fade away during the following two to eight weeks. Theoretically at this time the number and content of the red corpuscles is normal whereas the reduction in the number of white cells is marked so that one would expect a cat to be particularly susceptible to other infections at this time and yet I never recollect having a convalescent case develop any infection. Perhaps Dr. Gledhill could explain this "fading away" and also explain why these cats with such a markedly low white cell count do not contract other infections.

The clinical picture varies considerably according to the local conditions. In residential districts, where sporadic cases are more commonly seen than actual epidemics, diagnosis is often extremely difficult. Many cats over three or four years old appear to have some immunity to feline enteritis and the virus presumably lowers their resistance so that some other virus or bacteria can be established. We take the simple line of calling all such cases "cat 'flu" and

explaining to the owner that there are several different kinds and that the cat in question has a serious, medium or mild type. Otherwise I find myself explaining to a very disbelieving owner that cats with feline enteritis seldom have diarrhoea! Or that the cat has feline enteritis but that if it recovers it will have a permanent immunity and the owner replies that I said the same about the same cat last year! Probably the most constant symptom reported over the telephone is that "something has come over the cat's eye"—in other words the third eyelid is noticeable. Unfortunately this occurs whenever a cat does not feel well, but if accompanied by dullness in a young cat I advise immediate attention.

In more thickly populated areas in a town it is common to hear that "someone" has been poisoning the cats—usually on enquiry the deaths have all been in young animals and owing to the shortness of the illness poisoning has been presumed to be the cause, but as one or two older cats often become ill with what appears to be a subacute attack of feline enteritis I usually assume that this was the actual trouble.

All country veterinary surgeons will know the condition met on farms where the majority of cats die off and it is impossible to rear kittens. Presumably the disease is brought by a visiting tom; the cats are either seen looking mopey for a few hours prior to death or else are simply found dead. Any kittens introduced after this die off but a high proportion of town bred adults if introduced on to an infected farm will survive. If a female survives the original outbreak and breeds, the infection will be revived by each batch of kittens and may take a year or two to be eliminated. The period necessary for the infection to die out on a farm appears to be about six months but it is hard to get accurate and detailed evidence on such matters. One farm on which the disease occurred last autumn managed to save two young cats: one a female which very fortunately did not breed until late spring this year and there has been no evidence of illness among the kittens—showing, I assume, that owing to the age of the cat and the time of the year breeding was delayed until the virus had died. The average farm cat appears to have no resistance and so succumbs quickly and the outbreak is seen without the complications so common in town cats.

Post-mortem Examination.—I have no wish to add to nor to criticise the *post-mortem* results given by Dr. Gledhill as I have had very little experience. I do not like doing *post-mortem* examinations on such highly infectious bodies and I find that an owner who has taken the trouble to nurse a cat past the acute phase usually prefers to bury the body if the animal dies during the second phase. I had hoped to find, in Dr. Gledhill's paper, some help on the subject of the pathology of convalescent cases of feline enteritis.

Immunity.—The question of immunity as raised by Dr. Gledhill is interesting. In practice it appears quite straightforward. Those districts where the average dog of two to three years old is resistant to the canine virus diseases are the same districts from which adult cats may be taken and placed on infected farms without developing any symptoms of feline enteritis. Both species, I assume, develop an immunity through contact with repeated small doses of the virus. One rather curious case occurred the other day; a young female cat still suckling her 10-week-old kitten developed typical feline enteritis. The kitten was not kept away from her mother but as the milk dried up she ceased to suckle. Exactly 48 hours after the onset of symptoms in the older cat the kitten was taken ill and developed typical feline enteritis but in a much milder form—presumably the strain of virus was the same: so had the kitten some degree of resistance peculiar to itself or had it received some protection through the milk?

Differential Diagnosis.—Owing to the scarcity of specific symptoms in cases of feline enteritis differential diagnosis may be most helpful even if only in a negative manner.

(a) Other virus diseases. This is easy in the typical uncomplicated case but practically impossible if it is atypical or there is a mixed infection.

(b) Abscess formation. This condition is more common in male cats than in female or neutered cats; it is usually caused by the bite of another cat or of a rat. Careful examination should be made to ensure that there is no evidence of abscess formation on the body, legs or tail.

(c) Buried needles. I have twice put forward a tentative diagnosis of feline enteritis and subsequently found the cats had buried needles gravitating round the throat and shoulder regions. In neither case were the symptoms really typical of feline enteritis.

(d) Phosphorus. It is impossible in life to differentiate between phosphorus poisoning and feline enteritis. The severe liver disfunction produced by this poison causes a rise in temperature similar to that seen in feline enteritis. There is abdominal pain and death occurs in about 24 hours. Poisoning generally follows ingestion of the liver of a rat which has itself eaten poison—the phosphorus in the liver is very rapidly absorbed from the cat's stomach so that unless treatment is given within 20 minutes of ingestion there will be no phosphorus in the stomach and any subsequent vomit will not contain it. Many cats will not eat dead rats but a moribund rat would be just as dangerous.

(e) Tuberculosis. The onset of this disease is usually gradual, but when first called in to a case the history may be vague, there may be a rise in temperature, loss of appetite and abdominal pain. The course of the disease will vary and make the subsequent differential diagnosis quite simple.

Treatment.—Treatment, in my opinion, consists mainly of good nursing and prevention of secondary invaders. Routine treatment is penicillin or streptomycin and injections of either appear to cause clinical improvement and a reduction in the temperature. If penicillin is used there is often a stationary phase after the first two days of improvement but on switching to streptomycin the improvement is continued. I should be interested to know whether Dr. Gledhill considers that the antibiotics act directly on the virus or whether by reducing the number of secondary bacterial invaders they enable the cat to develop sufficient resistance to combat the virus as suggested by Dalling. The nursing consists of ensuring that the patient is getting fluid, warmth and comfort (in that order of importance). Siamese adults, in my opinion, are not constitutionally weaker than other cats but they do require more warmth when ill and all cats do best if nursed in some warm draught-proof place such as an airing cupboard. Dehydration is, I am sure, an extremely important factor in the course of this disease. If a cat can be persuaded to drink that is most satisfactory and the owners should be told to encourage fluid in-take of any kind. If, as usually happens, all food and drink is refused it is best to recommend spoon feeding, one teaspoonful every hour of water; water and glucose; milk, water and glucose or peptonised broth. Whole milk tends to lie in the stomach until vomited some hours later but the other liquids are usually absorbed if given in small quantities and the average cat does not bother to resist if only a teaspoonful is given at a time. If spoon feeding is impossible due to vomiting, temperament of the cat or temperament of the owners, then saline subcutaneously should be given once or twice daily. If the primary phase is survived it is wiser to warn the owner that the cat will be extremely delicate for some months. The exact pathology of this convalescent condition is apparently unknown at present but treatment as for liver and for kidney disfunction is most helpful in practice.

Prognosis.—Prognosis depends on so many factors that it is hard to make a definite statement. I am in agreement with Dr. Gledhill that there is a considerable variation in the virulence of this disease but whereas Dr. Gledhill assumes that this is due to a variation in the virus I am of the opinion that it is also due to the fact that many cats have some degree of immunity. Farm cats show little or no resistance to this disease, whereas healthy town cats over three years of age seldom die during an epidemic even if they do show symptoms. Generally speaking, a cat that survives the first 36 hours, if well nursed, has a very good chance of survival and of eventually making an apparently complete recovery but whether any permanent damage is left I do not know. It must be remembered that even if everything is in the cat's favour, if the cat has no will to live it is a waste of time to treat it.

Prevention.—Vaccine. A new vaccine for use in kittens or cats that may subsequently be exposed to infection has been produced and I have used this on three kittens. Twenty days after arriving on the farm the kittens were still very well and healthy.

Advice *re* infected farms and houses. This depends so largely on the method of spread of the disease, on whether recovered cats are carriers and on how long the virus can survive in buildings, that advice can be based only on practical observations. It seems advisable to keep a place clear for at least six months; all recovered females should be removed or their kittens destroyed at birth to prevent re-infection taking place.

Advice *re* breeding premises. As heard in Dr. Gledhill's paper,

disease spreads very rapidly; therefore it is wiser to isolate all new cats and cats returning from stud or show, in a separate building and it possible to have different staff attending the isolated cats. Information as to the incubation period seems vague but isolation should last 21 days.

The conscious thought which I have given to this disease during the few months which have elapsed since I was asked to open this discussion has shown me how much more clinical information would have been available if I had started "tidying up" my mind even a year ago. However, I see some eminent clinicians here to-day who I hope are also "tidy thinkers" so by the end of the discussion Dr. Gledhill should have a wealth of clinical material—and we hope, in return, that research on this disease will proceed more rapidly than it has done during the last two decades.

General Discussion

Miss Olga Uvarov (Ewell) congratulated Dr. Gledhill and Miss Brancker on their excellent contributions. The diseases of cats had been sadly neglected both by laboratory workers and clinicians and on reading through the available literature it appeared that since the days of the late Henry Gray very little work had been published in this country by clinicians on the problems of feline medicine. She felt that clinicians must be greatly blamed for this, perhaps more than laboratory workers. The cat problem had been taken for granted for far too long and when Miss Brancker said that tidy thinking was required the one animal which must have its diseases tidied up was the cat.

She did not pretend to criticise Dr. Gledhill's paper but there were one or two questions she would like to ask. Dr. Gledhill said on page 1 that coryza could develop during enteritis infection and she would support that on the clinical side. In these cases it was easy to find that there had been a complication, by the very characteristic changes in the mucous membrane of the eye and throat. To her mind these cats were carriers of the respiratory diseases, and she would like to ask Dr. Gledhill if he could say whether they carried both types of virus. He had described two types and she would like to know if any cats carried the two viruses.

She would also like to know a little more about the blood picture in this type of condition. The Americans had described it quite clearly; did Dr. Gledhill support that or had he found any other abnormalities? How quickly did the blood picture become normal in the recovered cases? Had Dr. Gledhill any cases of respiratory virus diseases in which feline enteritis had appeared as a secondary factor? Quite a lot had been said about the haemorrhages in the lymph nodes and she would like to know whether in recovered animals there was any appreciable clinical enlargement of these nodes? That might be of very great significance and interest in the middle-aged and old cats. One got a number of cases in cats with a very pronounced dyspepsia syndrome, where particularly the ileocolic glands were greatly enlarged, and she wondered whether these cats were cases of feline enteritis in the early stage?

One had to be grateful for the reintroduction of feline enteritis vaccine in this country. One had met cases in the Siamese where breeders had produced little bottles of American vaccine and one had felt a little bleak that one could only occasionally immunise Siamese cats and that it was not possible to immunise the ordinary cat. Now that the vaccine had arrived things would be very much better. She was glad Miss Brancker had not had her telegram to say that her experiments had relapsed.

She would like to know a little more about that characteristic symptom of feline enteritis, the pain. Could one say that the painful cry which the affected cat produced was localised to one particular organ or whether it was that the cat felt extremely ill and when the clinician was palpating, it gave this unmistakable cry? She thought most veterinary surgeons would recognise the cry without seeing the cat.

Miss Brancker had mentioned the obtrusion of the third eyelid; she could not support that, but she thought it appeared with any form of so-called cat 'flu in the secondary stage. If Miss Brancker saw this condition it was only when the cat was in the secondary phase of the respiratory disease or any other catarrhal complication. She was glad to hear her comments on treatment. Up to now this had been rather a hopeless disease and one had to be careful what one did. Antibiotics were a help in some stages, as were some of the sulphonamides.

From the point of view of administration of nourishment, a cat was a very tricky patient to nurse and she had found white of egg or concentrated protein was better than milk. She also found in dealing with a case of feline enteritis that one had to be extremely careful and gentle in handling the affected cat. The cat was in a fair amount of pain and unless one was careful it could be given a lot of unnecessary discomfort. One of the most important things, apart from warmth, was *rest*. Many people tried to do too much for the animal. A cat with feline enteritis must be treated twice as carefully as a man with a thick head.

Dr. Marcus Mason (U.S.A.) said that he had a predominantly small-animal practice and 25 per cent. of his clients brought cats. He had been very interested in this paper and was glad to see that work had been started to divide influenza into two virus diseases; much more should be done on these lines. It was a chronic disease and had a severe reaction and if one was trying to start a cattery it was not a question of 21 to 28 days but an indennite period because cats after this disease did poorly and were unable to rear good kittens for a long time. Drastic steps had to be taken to eliminate it and it was much more troublesome than cat distemper. The term enteritis was unfortunate because cat enteritis as such had hardly been touched upon. In the States the disease was referred to as feline distemper or panleucopenia in cats.

He thought the meeting might be interested to know something about his experience with the vaccine. He had used it for at least four and a half years and had had exceptionally good results. It was a formalised vaccine given in doses of 2 c.c., a week to 10 days apart. Protection was at least 90 per cent. He had raised some Siamese cats himself and had been interested in getting protection as early as possible at the least possible cost. He had tried to see how far down he could scale the dosage. The recommended dose was 2 c.c. but he got it down to 1 c.c. with good results. He tried $\frac{1}{2}$ c.c. but it was no good. In this vaccine there was a margin of safety of at least 1 c.c. He had tried intradermal vaccination, which he thought might give good results. He found that the serum from older cats or specific anti-sera had not been too successful. The course of the disease was very rapid and unless one had a large cattery and had to do something immediately he would not advise the use of serum, he would much rather use the vaccine in the cattery even where there were existing infections.

On the treatment of the cats or kittens where there was an epidemic or when one wanted to protect the kittens, the time to vaccinate was in the first two weeks after weaning. With a resident infection the mother cat did not necessarily infect the kittens—he thought there were other factors. He suspected fleas and ear mites carried the infection but that was something he had not been able to ascertain. He found a high incidence between fleas, ear mites and infection, and he strongly advised the use of vaccine as soon as possible. It was the same with dog distemper; one used to vaccinate dogs at four to five months, now it was done much nearer weaning time and the results were much better. As far as he knew there was no effective vaccine for cat influenza.

For the therapy of the cat distemper infection he had found in the last three years that the use of aureomycin and terramycin had reduced mortality from 80 to 90 per cent. to less than 50 per cent. Aureomycin was used first and very good results were obtained by using 50 mg. per day for three days. If it was used in higher doses or for longer periods the cat would develop diarrhoea and stomatitis as a result of the drug. One could produce this condition with this drug in a normal cat. Terramycin was much easier on the cats, they seemed to be able to take larger doses without any trouble, but he thought that aureomycin was the more effective of the two drugs. Against cat distemper penicillin and streptomycin were of little efficacy and where good results had been obtained he thought probably the diagnosis had not been very accurate, that the cases were mixed infections and what was being treated was probably cat influenza. His experience with cat 'flu was that the marked symptoms could be sharply reduced but with peniciilin or streptomycin the disease recurred as soon as therapy was reduced.

On another aspect of therapy of cat distemper, it was very important for these cats to maintain their water balance. They became dehydrated very rapidly and it was extremely important to spoon-feed them or get food into them somehow. He had had great success with amino-acids. One could give them by mouth; frequent mouth-feeding was very important. He did not believe in feeding every hour but he did believe in feeding three times a day. One must wrap the cat up so that it did not struggle, but it must be treated very gently and not allowed to get to the point where it would fight. Cats with cat distemper were very resentful, they would spit, gag and fight; a lot of really good nursing was involved but it was dangerous to over-handle them—he had seen cats killed by too much handling.

There was one other condition, possibly of virus origin, which had not been mentioned; he called the condition hyperpyrexia and it occurred in cats from two to four years old. They showed a very high temperature of 106° or higher, there was no vomiting, they refused food, they showed no pain of the characteristic type on palpation. He would put the cat into its basket and leave it and unless there was the weak cry which was a consistent clinical symptom of cat distemper, there was not much need for worry. The cats were usually in good condition; all they did was to refuse to eat, which was the symptom which brought the cat to consultation. At first he treated them with everything he had to get the temperature

down but nothing was successful and the cat seemed to go back to eating normally in its own time, the temperature dropping in three to four days by itself. He had not carried out any blood tests on these cats; he had not had enough cases to do so but he would put it forward for discussion as a possible variant of a known or perhaps new virus. In the untreated cases the mortality had been zero.

Mr. H. Raynor Hewetson (Southport) said he had not a great deal to add apart from congratulating Miss Brancker and Dr. Gledhill. He imagined his own thoughts were very untidy but one of the difficulties seemed to be whether they were all dealing with the same infection. Dr. Gledhill had gone to a great deal of trouble to find out where the disease was originally described and whether they were dealing with the same thing. The one thing which seemed to be constant was the symptoms. Miss Brancker had pointed to a significant factor when she referred to the different conditions in different localities.

He had found in other diseases, particularly canine distemper, that one got a different trend of symptoms and a different sort of mortality in different parts of the country. The predominating diagnostic note about the condition under discussion was in the early symptoms. Nobody had mentioned what he seemed to find, that the original vomit was just clear watery fluid and this was followed later by a bile-stained frothy fluid. Until the last 6 or 12 months he did not think he had seen diarrhoea associated with enteritis. One did see it now, but whether it was due to changing conditions or whether they were getting different strains of the virus one did not know.

On the question of mortality, he did not think that, however one treated the animals, the mortality ratio of the disease was anything as high as it used to be; he did not expect a mortality of 40 or 50 per cent. whatever treatment he used. When did a cat become immune, and did vaccine produce a lasting immunity which was worthwhile? In his opinion there was not much in age as far as immunity was concerned. He had had outbreaks in the cattery when the cats had ranged from 2 weeks to 10 to 12 years old and the immune animals had not been immune by age. In one outbreak 24 out of 27 cats went down, all of different ages. If vaccine was going to be used he did not think it was safe to assume that the cat would be all right when it was 12 months' old. He had frequently observed a single case in a cattery with all other cats remaining healthy. One might say that it was not feline enteritis but when one had gone through the typical symptoms he did not know what else it was to be called.

Another peculiar thing was that there were times when there was little apparent general infection, yet an animal which had a surgical operation would succumb a week later. Cats which had been castrated would come back with the symptoms of feline enteritis. Female cats that were spayed at times when there was no known infection would be all right if they were sent home the day after the operation was performed, but if they were kept in for a week they invariably went down with it. He did not know whether there was any relation between surgical wounds and the development of the infection. At one period for three years there was an outbreak in his cattery every year and in the third year he noticed that one cat had been in on every occasion and had never been ill and he thought that it must be a permanent carrier. He had never noticed the fading away after the illness which Miss Brancker had mentioned. His general experience was that if a cat had recovered after 10 days or a fortnight it appeared for all normal purposes as a normal cat.

He heartily agreed with Miss Uvarov and Dr. Mason that gentleness and care in handling were more important than any treatment; more cats were killed with treatment than died of neglect. He would not force or struggle with a cat to give it medicines, injections, or anything else; he preferred to leave it to nature. The majority of cats were quite amenable to reasonably gentle handling, and in addition to penicillin or streptomycin in the early stages he also gave liver extract. If one was going to treat a cat for enteritis everything must be done at once or not at all. If they were going to die they would do so within the first two or three days; if they got over that period they usually got better.

The intravenous injection of dextrose, if one could get the vein, showed a good response. Protein hydrolysate seemed to keep the strength up but if it was given intraperitoneally it was invariably followed by death. It was unfortunate that we had not available in this country the antibiotics about which Dr. Mason had spoken. He had heard on many occasions of the wonderful results which followed aureomycin in this condition.

[Addendum: Mr. Hewetson writes, on October 15th:—

"On my return from Harrogate on September 14th I found that a cat, 10 months old, in my cattery had developed symptoms of enteritis on September 10th, and had died on September 11th. It had been in 11 days and there were 24 other cats in, all apparently well.

"On September 15th I had a report that a two-year-old cat, which went home on September 10th, was normal until September 13th, when it commenced vomiting, and died on September 14th. Also, on September 15th a cat in the cattery, five years old, commenced vomiting and died on September 17th. On September 18th, five cats belonging to one owner were discharged, and on September 19th a call was made to one two years old, which commenced vomiting as soon as it got home, and died on September 20th. A kitten, three months old, which had shared the same pen and feeding dishes, was all right but was given feline enteritis vaccine on this day, and has remained perfectly well. On September 24th another cat of this lot, aged two years, commenced vomiting, but on September 25th had apparently recovered, and has remained well since. The other two cats, ages four years and two years, were given vaccine on this day and have remained symptom free, and these and the kitten are fit and well now.

"The remaining 18 cats in the cattery at the time of the first case have all been entirely unaffected, and no reports have been received of any cat going out before the first case on September 10th having any illness.

"On September 27th, when only nine cats remained on the premises, a two-year-old cat, which had been in since September 9th under treatment for an abscess, developed a catarrhal cold. On September 28th, another cat, six years old, which shared the same pen as another cat of similar age belonging to the same owner, which had been in since July 21st, also developed a catarrhal cold. These two cats were ill for about 10 days and recovered. None of the other cats, including the one in the same pen as one infected, ever showed signs of any illness.

"This factual report shows considerable variance from the views we expressed at Harrogate."]

Miss L. H. H. Phillips (Haslemere) asked Dr. Gledhill if he could enlarge on the incubation period because it seemed to vary from 24 hours to 23 days. She had a case recently of an owner who kept chinchillas and creams; she exhibited cats and was given a certificate for a cream to go to Brazil. That same evening she had a call from a well-known surgeon who had bought a kitten from the same litter the previous day. When she got there the kitten was purring, its temperature was slightly up, and it had vomited. It had all the characteristic pain of enteritis; the prognosis was that the kitten would be dead within 48 hours. The breeder was contacted and told that the certificate must be rescinded and that the other kitten could not go to Brazil; it was certain to come out with the infection. Nothing happened for 10 days and the kitten was all right. Just as the machinery started going again for exporting the kitten to Brazil, it was taken ill and eventually died. The rest of the cats which were all mixed up in the cattery did not get the disease.

What was the carrier position? Was it possible, as Dr. Gledhill said in his paper, that a litter had a certain amount of immunity from the maternal blood which would last until they were three months old and then possibly the weakest member of the litter would go down first? Could some tomcat strolling in the garden be the cause of the trouble, because no reason for the disease at all could be found?

She had not found that antibiotics were any use. The surgeon was keen on her using chloromycetin; she did not do so then but on the few occasions on which she had used it she had found it produced a very marked anaemia in a very short time. What effect would that have on the leucopenia of cat enteritis?

She agreed with all the speakers about the necessity for handling the cats extremely gently; in fact, she would say don't handle them at all. If fluid had to be replaced she preferred to do it by saline injections. She found most owners were extremely bad at handling cats. They tried to get food into them at every hour and the cats struggled and became exhausted. She felt that they were better left alone. She was a little puzzled about the mucous infection. If one had a kitten with feline enteritis which survived long enough to get to the stage of ulceration of the mouth she always found that it recovered. She always regarded ulceration of the mouth as occurring in feline enteritis and in the more commonly called cat 'flu. The animal she spoke of had reached that stage, had started to eat again, and then collapsed and died.

Dr. Mason spoke of the condition with a very high temperature. She had never had the pluck to leave them entirely alone but she would try to do so next time. She always thought she was curing them but perhaps she was not.

Professor C. A. McGaughey (Ceylon) said he had little to add to the discussion except to add his meed of thanks to Dr. Gledhill and Miss Brancker for their interesting papers. In the island of Ceylon they were very familiar with feline enteritis and he looked upon it as the most important disease of young leopards. Leopard cubs were brought from the jungle to the zoological gardens and when old enough they were exported; year after year young leopards died of enteritis.

In passing, he would mention to Dr. Gledhill, with regard to his doubts if the disease described as feline enteritis in wild cats had been transmitted to domestic cats, that in the *Veterinary Journal*, vol. 106 of 1950, it was stated that the virus from leopards survived in 15 per cent. glycerol for 18 weeks and was then transmitted successfully to young kittens. The average mongrel cat was extremely resistant to the disease; even young kittens from mongrel cats seemed to be resistant. He thought they must be carriers of the virus. The source of infection at the zoo at Colombo appeared to be mongrel cats which prowled around the cages of the young leopards.

The mention of pain by one of the speakers reminded him of the intense pain which these young leopards evinced; sometimes they were almost maniacal in their symptoms, throwing themselves all over the cage. It was curious that the lion, the only one of the wild felines which bred well in captivity, was the only feline in which feline enteritis had not been described. Gross, of the New York zoo, said that feline enteritis had been found in only one animal outside the felidae, and that was in the racoon.

He wondered whether people interested in the production of vaccines and sera might not contemplate the use of the leopard as a suitable animal for production of these products. Apparently the young leopard was quite susceptible to the disease. He had noticed that feline enteritis in domestic cats seemed to come in waves every three years or so in Ceylon, whereas the disease appeared among the young leopards in the zoo almost every year.

Dr. R. F. Montgomerie (Beckenham) said that he was interested in this question of pain. In feline enteritis there was a most marked peritonitis. It was for this reason that pain was so prominent a feature, careful handling of the abdominal region a necessity and intraperitoneal injection a danger. He was not going to adopt the suggestion that one of the larger "cats" be used for the production of vaccine. During the war years his colleague, Mr. MacIntyre, did actually see feline enteritis among the leopards in the London zoo. *Post-mortem* examinations were carried out; it was a typical picture and a large amount of material for the preparation of vaccine was available. Vaccine was in fact made, and used successfully for the protection of a number of animals in the zoo.

He was, of course, a little concerned when Dr. Mason suggested the use of a dose of vaccine less than that recommended. It must be obvious that the quality of vaccine depended primarily on the concentration of the virus in the tissue. They might trust the average laboratory to produce as many doses as was reasonably possible out of one set of tissue. It would be wrong to assume that in recommending a particular dose the laboratory had done more than err on the safe side and it would be thoroughly unwise to give less.

Dr. Gledhill had suggested that the only difficulty in producing serum was that of testing. It was quite possible to produce a high titre serum in the cat, but the trouble was that one got such a small volume. He was sure that serum had a place in treatment where an outbreak appeared.

A point which had been made in the discussion was that vaccine might be used in the face of an outbreak. There was very little good evidence on that point and yet perhaps sufficient to suggest that clinical trial was worth while. There was no more valuable instrument than a clinical thermometer in many diseases. If in an outbreak in a cattery one used a clinical thermometer and used it carefully because of the abdominal condition, one might use the vaccine for therapeutic purposes in early cases so detected, although originally it was produced as a prophylactic.

Professor W. L. Weipers (Glasgow) also thanked Dr. Gledhill and Miss Brancker for their interesting introduction to this subject. He, like Miss Brancker, stressed the economic importance of this disease in farm cats. He was also particularly interested in the epizootiology of cat enteritis and the paper had raised many interesting points on this aspect of the problem. Infectious cat diseases were described early in the century and it was many years before a satisfactory differential diagnosis had been worked out. In the early 'thirties Findlay's and Dalling's papers on the subject clarified the position greatly and he, the speaker, had accepted their basis of classification. There were many interesting problems in these epizootics, particularly when the disease was first introduced into a completely susceptible population. His own experience of the disease in the 1920s had suggested that, in some districts at least, there was evidence that the virus was finding a completely susceptible population and the mortality was very high indeed. Even in these days many veterinary surgeons recognised that there were two distinct clinical entities but the credit for their differentiation belonged to Findlay and Dalling.

In the discussion there had been little mention of the syndrome which was so frequently associated with infectious diseases of cats, that was, ulceration in the mouth. The speaker recognised as a distinct clinical entity the condition of ulcerative glossitis which was associated usually with very high fever and, in many cases, was quite indistinguishable from an early feline enteritis. In actual practice, if one discovered a case of this kind in the early stages showing lesions in the mouth and tongue, it was reasonably safe to give a good prognosis. Most of these animals recovered either with or without treatment and were very much better in a day or two; incidentally, on recovery they appeared to be immune to feline enteritis.

A great deal had been said about the importance of secondary invaders and the present scientific trend was to discount the importance of these organisms in producing clinical variations of the disease. He was satisfied, however, that in feline enteritis the secondary invaders were probably of paramount importance in determining the course of the disease. Dalling and his co-workers made it clear that uncomplicated disease caused by the virus alone was comparatively simple and he considered that many of the serious complications could be attributed to the presence of a haemolytic streptococcus. In outbreaks of the disease in Glasgow the speaker found that sulpha drugs or antibiotics were very beneficial in feline enteritis and in many outbreaks reduced mortality from about 90 to 10 per cent., provided treatment was administered reasonably early in the disease.

While appreciating Dr. Gledhill's diffidence in identifying feline enteritis with leucopenia, he would like to draw attention to the article by Jennings (of Liverpool) which appeared in *The Veterinary Record*, in which he was satisfied that a naturally occurring disease in the Liverpool area in cats, showing typical symptoms of feline enteritis, also showed evidence of panleucopenia. He quoted Mr. Lauder's work in Glasgow where white cell counts as low as 2,000 had been reported in cases of feline enteritis.

He had been looking for evidence of Baker's disease in this country ever since it had been reported in the United States but so far had failed to recognise any entity that might be attributed to this type of virus.

In practice he considered that the best contribution the veterinary surgeon could make to the control of infectious cat diseases was in their general management, particularly in housing. In the belief that respiratory disease in cats was associated with carrier animals, many veterinary surgeons modified their method of housing so that animals were kept in comparatively small groups. Once a group had been formed, these animals were kept as a unit and if no disease appeared within a short period, it was presumed that it was unlikely that it would develop. The presumption was that no carrier animal was present in this particular group. The speaker could not agree with this observation. He was satisfied that the respiratory type of cat disease could start *de novo* provided adverse conditions of housing were established.

The other explanation was that some animals harboured the virus and in adverse conditions of housing the virus developed pathogenic properties and an epizootic resulted. By supplying satisfactory housing conditions, one could completely rule out the apparent spontaneous development of this respiratory disease.

Mr. Hewetson had explained the frequency with which one got infectious enteritis after the administration of an anaesthetic in castration or after any other minor operation. The writer was satisfied that in practice this was a very important predisposing cause. One had only to look back in *The Veterinary Record* to find people describing a serious disease from which cats died seven or eight days after minor operations. Surprisingly few people attributed the disease to the possibility of infection in their own surgeries.

He was intrigued by the suggestion of ear mites carrying the infection. At one time he considered this a possibility in view of the persistence of feline enteritis infection in premises and also taking into account the difficulty which was often experienced in ridding a premises of ear mites. In managing the cattery it was important that care should be taken to prevent the spread of infection from one cat house to another and a technique involving almost aseptic surgical procedures was necessary to ensure the limitation of spread of disease. Staff could, however, quite readily be trained in a technique which could be reasonably fool-proof. By this system any cat that was admitted to the kennels free of infection would be returned home in the same condition. With cats admitted for boarding, it was doubtful if the attention involved in handling animals for minor ailments such as ear mites was justified. The respiratory disease of cats in kennels was now very much less frequent than previously. He did not consider that it did not occur but the spread of the disease could certainly be minimised by satisfactory housing.

In infectious enteritis he had found the antibiotics extremely useful and he was interested to hear that in the London area the results were not very good. In Glasgow the mortality in outbreaks had been cut very considerably but there was a possibility that different secondary invaders were involved in these outbreaks. At a committee on which he sat two years ago he was interested to hear one London practitioner objecting that penicillin was not and could not be beneficial against the disease caused by a virus.

On broad principles he found that feline enteritis was etiologically

and immunologically similar to distemper in dogs. One attack conferred a solid immunity while the respiratory diseases of cats were much more like influenza; he knew of cases in which a cat had acquired "'flu infection" two or three times within 12 months. He was extremely interested in the remarks of the speaker (Dr. Gledhill) that two viruses had been found in this country responsible for respiratory disease in cats and that these gave rise to two fairly distinct clinical diseases.

The Reply

Dr. Gledhill, in reply to the discussion, said he had a very long list of questions and perhaps he should have said that in regard to feline enteritis he felt something of a fraud inasmuch as he had very much less clinical experience than most of the speakers and he had not worked on this subject primarily. It had been encountered from time to time in the course of keeping cats in the laboratory. His strains of respiratory disease and enteritis had come from laboratory cats, either in the laboratory in which he worked or in other laboratories. Anyhow, it seemed that few of the questions were capable of an authoritative answer and so he would give his own views in regard to them.

As to the first question raised by Miss Brancker, that cats which moved towards recovery remained delicate and ill for a long time and seemed to fade away and go thin, he had no suggestion to offer as to why that might be.

With regard to the mucous membranes appearing pale, it was the belief of Hammon and Enders in the United States that the virus attacked the cells producing erythrocytes as well as those producing leucocytes and the consequent anaemia associated with the disease might be related to the mucous membranes becoming pale. It might be more in evidence in the less acute forms of the disease.

Secondly, Miss Brancker asked why cats with leucopenia did not contract other diseases and he thought the answer was that they did contract other diseases. Many speakers had stressed the importance of secondary organisms and presumably their comments arose from the fact that the normal body defences were inadequate owing to the leucopenia. It was also to be noted that leucocytosis sometimes followed upon the leucopenia quite rapidly, at least according to Pavilanis, such leucocytosis having been stimulated by secondary infection with bacteria.

Miss Brancker raised the point, and nearly every speaker had mentioned the question, of whether there were carriers. It appeared that there was no scientifically controlled work which established carriers in feline enteritis, but it would seem almost impossible to explain the immunity of cats if one did not postulate that there were carriers. He had himself experienced a rise in immunity in cats when held for a long time in his own establishment. A similar observation had been made by many others who had worked with the disease. If they kept cats any length of time they were almost invariably immune, whereas if they drew them from a private source there was a good chance of their being susceptible. Observations such as these strongly suggested both that there were carriers and that immunising infections without overt symptoms were common. The matter required more scientific demonstration.

In the case of the respiratory diseases, Baker recovered virus from animals for some time after recovery from feline pneumonitis. The fact that the immunity of recovered cats was by no means perfect in either of the respiratory diseases would lead one to expect that the viruses might persist after recovery, especially as recovering animals often had relapses.

Miss Uvarov had raised a number of interesting questions but there was no answer to them as yet.

He was very pleased to hear Professor Weipers mention that cases of leucopenia had been found around Glasgow. No doubt he (the speaker) had overlooked the contribution in *The Veterinary Record* on the matter.

The reason for the little attention directed towards research into the diseases of cats was related to cats being of small economic importance. Funds for research were mostly provided directly or indirectly by the Government and were therefore less readily available for cat research than for research on diseases of animals of greater economic importance. On the other hand, great impetus to research on virus diseases had followed upon the work of Laidlaw and Dunkin on dog distemper.

Miss Uvarov and Professor Weipers had noted that the respiratory diseases might occur secondarily during the course of feline enteritis and they wondered whether feline enteritis was ever secondary to the respiratory diseases. In passing the respiratory viruses experimentally, feline enteritis had appeared on several occasions, but it had seemed more likely that such cats had been infected with material contaminated with feline enteritis virus rather than that they were themselves carriers of it.

He was specially interested in what Dr. Mason had said about conditions in America and to learn what success he had had with antibiotics. With regard to the hyperpyrexia he mentioned, he could not say that he had encountered any such condition except on one occasion with a group of cats which had been used for work on respiratory diseases. Before finally disposing of the cats he thought he would challenge them with feline enteritis virus. Out of 30 cats three died of feline enteritis. Ten others showed temperature responses which were quite sharp although no other symptoms were apparent. The remainder were quite unaffected by the challenge. Thus infection could cause a sharp temperature response which was incapable of being diagnosed, at least by ordinary examination.

The question of incubation was important from a practical point of view. It had been stated to be about two to nine days, although there were some differences of opinion, two to six days and five to eight days being also given. These differences of opinion might in part be due to the means used for judging when cats become ill. The first temperature rise and indications of leucopenia occurred 24 hours or more before symptoms were obvious. Then, again, it might be that the incubation period varied with different strains, just as two strains of Hindle and Findlay, mentioned in the paper, gave rise to diseases of duration differing by more than 12 days.

Sheffield University Investigation into Toxoplasma Infections

The Chairman then called upon Dr. C. P. Beattie, Professor of Bacteriology in the University of Sheffield, who said that the Council had very kindly allowed him to ask for help in an investigation which he and a colleague were carrying out on toxoplasma infections. This was one of the most successful parasites in that it affected nearly all species of birds and mammals and rarely did it produce any disease. If that were all he would hardly be addressing the meeting, but it did sometimes produce serious disease in man and animals. When it appeared in rabbits their spleen and liver were enlarged and there were small tubercle-like nodules in the liver. In dogs it produced a disease rather like distemper, associated with emaciation, diarrhoea, and ulceration of the intestines. In the human being the disease appeared in the infant which was born with hydrocephalus, was often mentally backward, had convulsions or other signs of disturbance of the nervous system, characteristic eye changes and frequently intracranial calcification. It had sometimes been seen in the adult where the disease resembled typhus, encephalitis, or glandular fever.

The particular interest of Dr. Beverley and himself was the question of where the infection came from. The infection of man must come from some other source and it seemed likely that it might come from animals; if so, what were the animals? A case was discovered in a butcher's boy, and then it was found that his employer also gave a high titre antibody reaction. This led them to think that it might come from meat. An investigation was carried out to look for antibodies in workers in slaughterhouses and meat markets. Skin tests and tests for serum antibodies were carried out. The results showed that whereas 36 per cent. of people handling cattle, sheep and pigs gave positive results at a titre of 1/4 or more in the "dye" test for toxoplasmosis, little greater than the general adult population (28 per cent.), rabbit handlers gave a much higher percentage of positive reactions (64 per cent.). Rabbits might be one source. Other likely sources were the common domestic animals, dogs and cats.

There had been a most interesting discussion about cat diseases and he knew that there were many present who dealt with cats or dogs and he was asking for their help by allowing him to test a small sample of their serum and to carry out a skin test to find out how the percentage of positive reactions among veterinary surgeons compared with the percentage in the general adult population and in slaughtermen and rabbit handlers. He would also be very grateful for any help which veterinary surgeons could give him by sending specimens of blood from dogs and cats with symptoms suggestive of toxoplasmosis.

[Thirty-one veterinary surgeons very kindly gave specimens of their blood and allowed skin tests to be done on them. The results of the Sabin-Feldman "dye" test for toxoplasma antibodies showed that 35 per cent had antibodies active in a titre of 1/4 or more. This is almost the same as the percentage found in slaughterhouse workers and little greater than in the general adult population (28 per cent.). It is much less than that found in rabbit handlers (64 per cent.).

Positive skin test results agreed well with positive "dye" test results at a titre of 1/4 or more. There was only one discrepancy, where a negative skin test was associated with a "dye" test of 1/24.

Grateful thanks for their help are offered to the B.V.A. Council and to the participating volunteers—C. P. B.]

The Chairman proposed a very hearty vote of thanks to Dr. Gledhill and Miss Brancker and all those who had taken part in the discussion, helping to elucidate the position as far as possible with regard to the disease of feline enteritis.

TRYPANOSOME INFECTIONS OF CATTLE—DIAGNOSIS BY BLOOD EXAMINATIONS WHEN PARASITES ARE RARE

BY

R. N. T.-W.-FIENNES,

East African Veterinary Research Organisation

Certain recent criticisms on work done by the writer were based on attempts to diagnose trypanosome infections in cattle by examination of thin blood smears. It was, therefore, decided to assess the relative values of thick and thin smears of blood for this purpose. The thick smear is the standard method used for this purpose by the writer and 600 fields are examined before a negative result is accepted.

For this purpose, blood was delivered from a pipette on to 10 microscope slides and the African assistant normally responsible for making these slides, a man of more than 10 years' experience, was instructed to indicate the quantity of blood that normally he would use. It was found that he required a quantity of blood in excess of 0·1 c.c., which he spread in a rough circle of diameter 2·4 cm.

The writer then estimated—very generously—the appropriate quantity of blood required to make a thin smear and calculated the area over which this was spread. Results were as follow:—

Thin smear :

Quantity of blood taken	1/200 c.c.
Remaining on spreader	At least 50%
∴ Actual amount of blood spread ...	= 1/400 c.c.
Area covered 2·5 cm. × 6·0 cm.	= 15 square cm.
∴ Thickness of smear in terms of wet blood	= 1/6000 cm.

Thick smear :

Quantity of blood taken = <0·1 c.c., the excess over this figure will cover that left on the spreader, so that a figure of 0·1 c.c. may be taken as correct.

Radius of blood circle	= 1·2 cm.
Area covered	= 4·9 square cm.
Average thickness of blood	= 1/49 cm., say 1/50 cm.

We, therefore, find that thick smear examination is 120 times more effective than thin smear examination. This is to say that to equal the results of examination of 600 fields of a thick smear, it would be necessary to examine 72,000 fields of a thin smear.

Using X5 eyepiece and X100 objective, the area of each field of the microscope was calculated from measurements obtained with a stage micrometer. By this means, it was calculated that, using the thick smear method, at least 2·4 cubic mm. of blood were examined in 600 fields. Thus, if daily examinations are made on six days each week, as much as 14·4 cubic mm. of blood will be examined. This should give every chance of detecting trypanosomes, if present in the blood. Nevertheless, the scanty counts of parasites that sometimes occur reveal how essential it is to make regular examinations by the thick smear method. Some recent counts, selected at random from recent work, are given in the table below to illustrate this point.

Table

Showing Trypanosome Counts in Calves using the Thick Smear Method. Figures indicate the Number of Parasites seen in 600 Fields

No.	5.5.52	6.5.52	7.5.52	8.5.52	9.5.52	10.5.52	12.5.52	13.5.52
286	—	—	—	—	—	—	—	—
287	TC 1	—	TC 3	TC 1	—	TC 2	TC 2	TC 1
295	TC 2	TC 1	TC 3	TC 2	TC 1	TC 2	TC 3	TC 1
298	TC 1	—	TC 2	TC 2	TC 1	TC 1	TC 3	TC 1
299	TC 3	TC 1	TC 3	TC 2	TC 1	TC 2	—	—
300	—	—	—	—	—	—	—	—
301	TC 3	TC 2	TC 3	TC 4	TC 2	TC 3	—	—
302	TC 2	TC 1	TC 2	TC 2	TC 1	TC 2	TC 2	TC 1
304	TC 2	TC 1	TC 3	TC 2	—	TC 1	TC 1	TC 1
305	—	—	—	—	—	—	—	—

TC = *Trypanosoma congolense*

It is evident that the thin smear method could only fortuitously have revealed infection in the cases quoted above.

Summary

A comparison is made between the thin and thick smear methods of diagnosing trypanosomiasis in cattle, where parasites are rare in the blood. It is concluded that the thick smear method is at least 120 times more effective than the thin smear method. The thin smear method is not effective in detecting scanty infections.

INTERNATIONAL WILDFOWL ENQUIRY COMMITTEE

Duck Adoption Scheme

The fourth annual report on the duck adoption scheme indicates that, this year, a pintail duck has wrested the long-distance palm from the widgeons, which had retained the honour since the scheme was instituted four years ago. This duck, however, achieved nothing like the distance covered by the widgeon that flew from Britain to Siberia a year ago. Flying under the patronage of the R.A.F.—in the guardianship of Mrs. Buchanan, the wife of an officer in the service—the pintail reached latitude 67 deg. 50 min. N. in Finland, well over 1,000 miles from the station at which it was ringed. just outdistancing two unadopted drake teals which were shot across the border in north Sweden.

The report notes that there were far more travellers to the south than usual. One of them, a teal duck, reached the mouth of the Guadalquivir, in Spain, the farthest flight in that direction yet recorded by an adopted bird. This teal is owned jointly by Mr. W. B. and Mr. F. A. Leach, of Newmarket.

Once more, between 2,000 and 3,000 ducks and geese were ringed during the season at the English and Welsh stations of the International Wildfowl Enquiry Committee, which runs the adoption scheme as a means of raising money to maintain its decoys. For a payment of 5s. anyone may adopt a duck carrying a numbered aluminium ring and will be kept informed of any news that may come in about its movements. The address of the Duck Adoption Scheme is care of the British Museum (Natural History), Cromwell Road, London, S.W.7.

* * * * *

The National Stud has purchased the stallion Tenerani from Signor Teslo for the sum of £20.000. Tenerani. which won the Queen Elizabeth Stakes and Goodwood Cup In 1948, has been on lease to the National Stud during the past year.

CLINICAL COMMUNICATIONS

TREATMENT OF PREGNANCY TOXAEMIA IN EWES BY ORAL ADMINISTRATION OF GLYCEROL

WYN R. GRIFFITHS, F.R.C.V.S.,
NEWPORT, MON.

History.—Outbreaks of pregnancy toxaemia on two farms were attended and treated. Flock A consisted of about 80 cross Clun ewes of ordinary type. These had been kept on bare grass with no supplementary feeding; they were moved to another farm and feeding of hay commenced on February 20th, the day the first cases were reported. Thereafter the flock was inspected four times daily at 7 a.m., 11 a.m., 8 p.m. and 6 p.m. Eight of the 12 cases treated were in very fat condition in the autumn, but had lost a considerable amount of flesh during the winter.

Flock B had suffered great losses in the past; last year about 7½ per cent. were slaughtered a day or two after symptoms were seen, since previously no cases had recovered. This flock of about 1,000 ewes was brought into spacious lambing fields and given mangolds in addition to the ample grazing available.

Diagnosis.—The disease was typical enough to be diagnosed clinically. It was not possible to take blood samples from all cases, but those analysed showed the presence of acetone, which was to some extent confirmatory. It was intended to take samples after treatment, but recovery was so rapid and complete that it was very difficult to catch the ewes again and facilities at the first farm were not ideal for the purpose.

Treatment. — Treatment usually is considered useless. Nevertheless, because it is well known that disturbance of carbohydrate metabolism is a feature of this disease, and because glycerol can enter into the carbohydrate cycle and be converted into glycogen and glucose, it was decided to try oral administration of glycerol for therapeutic purposes. Glycerol was given *per os* in doses of 4 oz. mixed with equal quantities of water with some chlorodyne added. The results were remarkably good: in many cases ewes previously affected were grazing in about one hour, while those severely ill and recumbent on their sides were able to walk in one and a half to two hours. Relapses sometimes occurred but these responded to further therapy. The mixture was administered warm in order to pass straight to the abomasum; thus recovery was hastened. Recovered animals showed great thirst for several days to correct the dehydration incurred. One ewe suffered several relapses but responded each time to treatment. She lambed two very good lambs 26 days after the first attack and seven days after the last. This ewe probably was suffering from a lowered protein reserve and would have benefited from good protein feeding.

The addition of a small quantity of weak copper sulphate solution would hasten the entry of the mixture into the small intestine for absorption, under conditions when the warming of the mixture would prove difficult. The mixture proved very palatable, ewes requiring a second dose taking it readily.

The necessity of early diagnosis and immediate treatment, before severe liver damage is sustained, cannot be emphasised too strongly.

CASE REPORTS

FLOCK A

No. 1.—February 20th. Ewe had been ill about four days. Recumbent; very strong smell of acetone in the breath. Given 8 oz. of mixture; found dead next morning.

No. 2.—Found ill in field, February 20th. Smell of acetone in the breath. Treatment followed by recovery; relapsed and treated again; recovered on February 23rd. Lambed February 26th; three lambs born alive. One, very weak, soon died; the other two were normal and were reared.

No. 3.—February 21st. Seen twitching eyelids and ears, smell of acetone in the breath. Treatment followed by rapid recovery. Relapsed, very ill, down in brook on February 23rd; recovered after treatment. Lambed two live lambs on March 1st.

No. 4.—February 23rd. Found ill, treated, recovered. Lambed March 9th, two live lambs.

No. 5.—Found ill first on February 23rd. Relapsed four times; mild symptoms seen on each occasion. Treated and recovered on February 23rd, 29th, March 7th, 9th and 13th. Blood sample taken on March 13th :—

Calcium	...	10·3 mg. per cent. serum
Magnesium	...	2·1 " "
Acetone	...	15·0 " "

Smell of acetone in the breath with each attack. Lambed March 20th, two live lambs.

No. 6.—Treated February 28th, followed by recovery. Lambed March 13th, two live lambs.

No. 7.—Ill March 2nd. Recovery after treatment; relapsed March 7th; treated and recovered. Lambed two live lambs March 16th.

No. 8.—Ill March 2nd. Treated and recovered. Lambed March 13th, two live lambs.

No. 9.—Ill March 4th. Treated and recovered. Relapsed March 9th; very severe. Treated and recovered in one and a half hours. Lambed March 10th, two live lambs.

No. 10.—Ill March 8th. Treated and recovered. Lambed March 10th, two live lambs.

No. 11.—Ill March 9th. Treated, partial recovery; treated again March 10th; complete recovery. Lambed March 21st, two live lambs.

No. 12.—Very mild symptoms March 16th. Smell of acetone in the breath. Blood sample taken :—

Calcium	...	10·0 mg. per cent. serum
Magnesium	...	1·8 " "
Acetone	...	20·0 " "

Treatment followed by recovery. Lambed March 17th, two lambs: neither survived; ewe cleansed normally, but died March 19th. It was not possible to attend this flock every day, hence the omission of the smell of acetone in the breath as a symptom in many cases as the owner was unable to appreciate the odour.

FLOCK B

No. 1.—Six-year-old Welsh mountain ewe in fat condition. Attended March 20th. Very ill and recumbent on its side; probably ill about three days. Limbs quite limp. Strong smell of acetone in the breath. Treated; one and a half hours later unable to rise. Placed on its feet, walked 40 yards, stumbled and fell; was picked up, and walked another 60 yards. Left sitting comfortably on its brisket. Blood sample taken :—

Calcium	...	9·5 mg. per cent. serum
Magnesium	...	2·2 " "
Acetone	...	35·0 " "

Treated again same day, March 20th. Found dead next morning.

No. 2.—Very severely ill on March 20th, with strong smell of acetone. Ill about three days; similar to Case No. 1. Treated and found normal next morning. Blood sample taken :—

Calcium	...	9·3 mg. per cent. serum
Magnesium	...	2·5 " "
Acetone	...	40·0 " "

Lambed March 24th, two lambs, both dead. Ewe suffered from dystokia; lambs delivered manually by owner and ewe died two days later.

No. 3.—Ill March 21st, smell of acetone in the breath. Staggering about; no difficulty to catch. Treated and recovered. Blood sample taken :—

Calcium	...	10·8 mg. per cent. serum
Magnesium	...	3·4 " "
Acetone	...	20·0 " "

Two live lambs, March 30th.

No. 4.—Wiltshire horn half-bred ewe. Ill March 22nd. Treated and recovered; relapsed March 24th, with mild symptoms; treated and recovered. Lambed April 1st, two live lambs.

SUMMARY

Outbreaks of pregnancy toxaemia in two flocks of ewes are described.

In one flock 11 out of 12 cases and in the other three cases out of four recovered after treatment with 4 oz. of diluted glycerol.

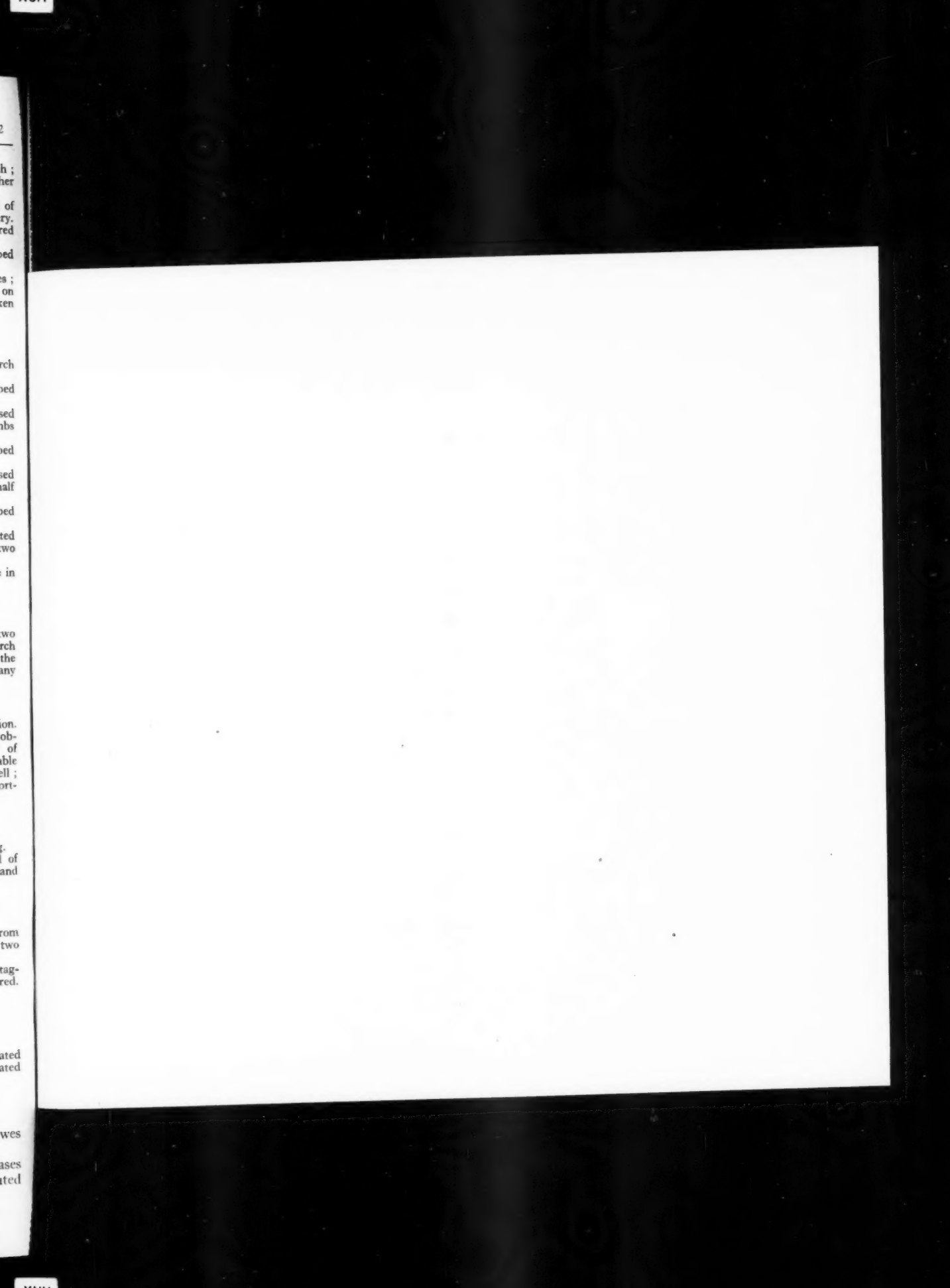

FIG. 1.

FIG. 2.

Acknowledgments.—The writer is grateful to the Veterinary Laboratory of the Ministry of Agriculture at Weybridge for examination of the blood samples.

* * * * *

INJURIES TO THE STIFLE JOINT IN YOUNG DOGS

A TRAP FOR THE UNWARY

N. ROBERT TURNBULL, F.R.C.V.S.,
BASINGSTOKE

Young dogs up to the age of about 12 months frequently injure their stifle joints when playing roughly or galloping about on uneven ground.

The injury is characterised by the limb being held in flexion, and on manipulation of the joint there is a severe pain reaction. The injury can vary in severity from a simple sprain to a separation of the distal epiphysis of the femur. The severe pain not infrequently renders an accurate diagnosis impossible without the use of X-rays. It is in the interpretation of the X-ray picture that one finds the " trap for the unwary." There can be no mistaking the separation of the epiphysis, but the appearance of the normal tibia in a puppy up to the age of 12 months shows an apparent fracture of the crest of this bone. This is due to the fact that the tuberosity of the tibia has a separate centre of ossification, and does not become fused with the rest of the bone until after 12 months.

The majority of stifle injuries in young dogs are merely severe sprains, and recovery is usually complete in three to seven days, and one should not be led into mistaking the normal appearance for a fracture.

The attached photographs will illustrate these points.

Fig. 1 shows a normal joint as described. The arrow indicates the as yet unfused crest of the tibia.

Fig. 2 shows a separation of the distal epiphysis of the femur, but the crest of the tibia presents the same appearance as in the normal joint.

I submit these observations in the hope that they may help others to avoid the trap into which I fell before I acquired familiarity with radiographs.

QUESTIONS AND ANSWERS

The submission of questions for inclusion in this column will be welcomed; they can relate to any aspect of veterinary work, but must be of a general nature—questions cannot be considered in which information is sought regarding diagnosis or treatment of specific cases, nor for which solely a private reply is desired. For purposes of record, each question is numbered, and those submitting questions are required to furnish (not for publication) name and address.

Answers to readers' queries represent exclusively the personal opinions of the writers, and criticism of the replies will be appreciated.

All communications should be addressed to the Editor.

Intermittent Diarrhoea in a Cow

Q. 418.—*An eight-year-old pedigree Friesian cow, since last December, has suffered from attacks of diarrhoea at intervals of about six weeks. The diarrhoea is evil smelling, and is accompanied by loss of milk and condition and subnormal temperature. Recovery takes place in about two to three weeks, with or without treatment. Numerous faeces examinations have proved negative. Any helpful comments, please?*

A.—The case described is almost certainly associated with a chronic affection, and probably infection of some part of the intestinal tract. By far the most likely cause of this train of symptoms is Johne's disease. Failure to find organisms in numerous samples of faeces does not rule it out. The writer took 11 samples from one cow before organisms were found. Mr. N. H. Hole, B.Sc., M.R.C.V.S., of the Pathology Department, Laboratory of the Ministry of Agriculture, Weybridge, will carry out a complement fixation test on a sample of 2 c.c. of clotted blood. The writer understands that this test is still in the experimental stages, though a great many such tests have now been carried out and a positive finding in such an animal would greatly increase one's suspicion. Other chronic infections of the alimentary tract include *F. necrophorus* and *Actinobacillus lignierisi*, but in the writer's experience neither of these sets up diarrhoea. Any other cause must be a matter for speculation. Such a case is always unsatisfactory and worrying to the clinician, and generally ends in slaughter.

* * * * *

Ulceration of the Vulva in Bitches

Q. 419.—*What is the cause and treatment of a particularly stubborn form of ulceration of the vulva in bitches?*

A.—The respondent is not aware of any specific form of ulceration of the vulva in bitches. One case seen was attributed to some hormone deficiency and a course of thyroid extract together with local application of a benzocaine ointment proved successful.

Some bitches have a vulva which is very much recessed in the surrounding folds of skin and such animals are more liable to ulcerative conditions ; ordinary hygienic measures by the owner are usually enough to promote healing. Other bitches tend to produce excessive quantities of a ceruminous material around the vulva and this mats the hair and may lead eventually to a condition similar to that seen in ceruminous and hairy ears. Cleansing and the application of a dressing containing salicylic acid are helpful in such cases. Useful salicylic acid preparations are lin : salicyl : co. or conspersus ac. salicyl. co.

As the condition appears to be of a secondary nature, due to a primary irritation or disease, other possible causes are : (1) Extension of pruritus ani, and/or anal eczema. (2) Anal abscesses. (3) Foreign bodies, such as grass seeds. (4) Excessive acidity of urine, as revealed by a urine examination. In this condition two bitches, by licking and biting, have been observed to produce ulcerative lesions of the vulva.

* * * * *

CORRESPONDENCE

Restraint of Bulls

Sir,—In connection with the item relating to the above (Q. 416), in No. 39, Vol. 64, page 581, of *The Veterinary Record*, we venture to inform you of the following experience. Eighteen months ago one of our valuable A.I. bulls pulled his ring through septum and nostrils, the result being a gaping wound. Realising that this bull, without a good ring, would be useless for our bull station because of his temperament, we decided to stitch the wound.

Using ropes, the head of the bull was immobilised as well as possible between the poles in the shed, as was his body, to some degree, with heavy ropes.

The use of local anaesthetic appeared to be impossible because of the extraordinarily tough tissue of the nose ; therefore, we anaesthetised the surrounding area, especially the trigeminal nerve, with conduction-anaesthesia.

The edges of the wound were freshened and the wound itself was disinfected with penicillin.

The great resistance of the tissue to the entry of the suture needles had to be overcome by force. We used heavy and medium needles of the usual type, cutting-edged, bent, and with an open eye. Two sutures with heavy silk brought the edges of the wound into the right position. After this about six sutures with medium silk were inserted, so that all the accessible edges of the wound were closed.

The bull resisted strongly, especially at the time of anaesthesia, but thanks to the excellent degree of immobility we could control the animal sufficiently and there was no real difficulty or danger.

Without further treatment or special precautions, the result was then awaited, the bull being tied up by the head : he was looked after with great care.

After eight days the sutures were removed. The wound showed primary intention healing, and after six weeks the new ring was inserted. The small white scar line is now practically invisible and the animal is even slightly easier to handle than he was before the accident.—Yours faithfully, G. GROOTENHUIS, Sub-Director, Animal Health Service, Zeeland, The Netherlands. *October 22nd*, 1952.

MEMOIR

JAMES THOMAS EDWARDS, D.Sc., M.R.C.V.S.

Scientist, Teacher, Editor and Historian

This week we are called upon, with tragic unexpectedness, to pay tribute, which we hope will be amplified and made more worthy by the publication in later issues of personal appreciations, to one whose devotion to the profession, to the British Veterinary Association and to this its weekly journal, was so liberal in its outpouring as to command the homage of us all.

It is but a short while since the belief that Dr. J. T. Edwards would emerge safely from his recent serious illness was shared by all his friends—and how countless they are, from his revered continental savants to the latest Camden Town School recruit to the ranks of the Central Veterinary Society, in whose company he passed his last, happy evening on Thursday, November 6th. It will be recalled that barely 12 months after the Association, at Eastbourne, had bestowed upon him the highest honour in its gift, in the conferment of Honorary Membership, he was struck down with double, apical pneumonia. For weeks he lay in University College Hospital, whither he had been taken after he had been found, at death's door, in his lodgings in Canonbury, where with the common indifference of genius to physical discomforts he had lived since the temporary closure of his normal residence, the University of London Club. Coronary thrombosis was the grim legacy, but even then all might have been relatively well for a space, had he not turned a deaf ear to the pleas of his medical advisers and other friends and refused to "take things quietly." It was, indeed, characteristic of him that, with or without good reason, he felt he must be at the centre of things: such was the measure of his tremendous, overriding concern for the well-being of his profession, corporately and as individuals, as he saw it. Attention to everything except his health—to his writings, his researches into veterinary history, and his concern with our current "professional politics"—was imperative to him, and we cannot but feel that his passing was as he himself would have wished, but for the agitation caused to others: to sink peacefully into unconsciousness in the 'bus that was taking him home from his meeting with fellow members of the Society so dear to his heart and which he ever regarded as the choicest of the many venerable links in the Divisional Chain of our Association.

As we reveal below, and as others we trust will testify from their fuller knowledge of particular phases of a remarkable career, Dr. Edwards rose to great heights in his profession and achieved great things. For ourselves, we confess that often we encouraged that readiness of his to absorb himself in professional interests that were not concerned solely with his work: yet how irresistible was that quite unique zeal to help, in any capacity, at any time, in meeting an editorial need! In those early days of Midlands publication, Dr. Edwards, our first editor (under the N.V.M.A. regime), was wont, after an arduous day's teaching at the College, to make a desperate, often unavailing, dash with the following week's "make-up" of the *Record*, due for printing next morning, to catch the midnight mail to Birmingham; or, in much later years, and greatly to the annoyance of the resident caretakers, he would encamp for months at Gordon Square while pursuing his day and night onslaught on his mammoth contributions to one of our special numbers. Whatever supporting pillars of spontaneous readiness to assist them other periodicals may find, we, at least, shall not look upon his like again.

A man of great personal charm and depth of feeling Dr. Edwards was ever the good companion—earnest or gay as occasion required. His contributions to political debate or scientific discussion were emphatic in style: the former gained the respect accorded to conscientiousness; the latter carried the conviction of an accepted breadth and depth of knowledge and experience.

. . . . . .

James Thomas Edwards was born on November 5th, 1889, at Kidwelly, Carmarthenshire, S. Wales, and, as he was fond of recalling with pride as evidence of a sturdy "kinship with the soil," his childhood and early life was spent in farming conditions. He had all the quick wit, native intelligence (and volubility!) of the Welsh, and when he went to the London College, in 1907, he took every available R.C.V.S. prize. He qualified M.R.C.V.S. in 1911 and, also as a matter of record, we may add here that he took his B.Sc. in Veterinary Science at the University of London in the following year and his D.Sc. in 1926, the subject of his thesis being "The Chemotherapy of Surra in Horses and Cattle in India." The year 1911 was a momentous one for James Edwards, for after a brief spell in general practice, notably with John Campbell Hill, of Llanelly, whom and whose family he held in lifelong affection, he was appointed Junior Research Assistant to Sir John M'Fadyean in the Research Department of the Royal Veterinary College. Shortly afterwards he obtained a research scholarship, tenable for three years, in veterinary pathology. Of this time, one year (1912-13) was spent in Paris, where he underwent a

course of training at the Pasteur Institute and carried out some research work under Professor Vallée at the Alfort Veterinary School and formed those friendships with Professor Leclainche and Dr. Ramon he valued so highly in later life. The remainder of his tenure of the scholarship he spent with his own great master of Pathology at Camden Town. There he also prosecuted research into Johne's disease and vaccinated cattle against tuberculosis, publishing papers on this work, in collaboration with Sir John and Dr. Leslie Sheather, in the *Journal of Comparative Pathology and Therapeutics*, of which quarterly, on the death of the former, he became joint editor.

In 1914 some researches of his into canine distemper sustained the universal interruption, and it was with the promptitude born of a vigorous and idealistic temperament that he joined the Royal Army Veterinary Corps at once. In August, 1914, therefore, J. T. Edwards went to France with the Expeditionary Force, serving mainly with the Royal Field Artillery and gaining the Mons Medal, with Bar, and being mentioned in despatches (November, 1915). With the rank of Captain, he was recalled from Army service in June, 1916, to continue work at the Royal Veterinary College, this time in connection with contagious abortion of cattle, but also working with Sir John M'Fadyean on research into contagious abortion in mares and joint-ill in foals. As Senior Research Assistant, Edwards carried out the duties of demonstrator in pathology and lectured in veterinary protozoology. When, at the Royal Veterinary College in 1920, the hitherto basic and, as it had been deemed, all-sufficient subject of stable management burgeoned into veterinary hygiene and was accorded a special Chair, Dr. Edwards was the first incumbent. He was an enthralling, but not a practical, teacher, for, as was the tendency throughout his life, he found in this as in every subject that interested him, an inexhaustible theme of discourse—seemingly oblivious of the fact that the date of Examination Day just would not allow of the pursuit of knowledge far up every avenue collateral to the syllabus.

India

In October, 1921, after five and a half years on the staff of the Camden Town School, there came a radical change of course in his career. In that month Dr. Edwards secured appointment as Director and First Bacteriologist of the Imperial Bacteriological Laboratory of Mukteswar, India (now the Imperial Institute of Veterinary Research). Here he was charged with the duty of manufacturing sera and vaccines for combating the serious cattle diseases of India, notably rinderpest, endeavouring (on the instructions of the Government) to place the laboratory on a self-supporting basis. As all who really knew him could have foretold, business proved not to be his "strong suit." Here, however, he carried out many activities of great value, such as training Indian personnel and conducting courses of post-graduate study for R.A.V.C. officers and men of Civil Departments, in addition to his main work. This was the immunological study of rinderpest. Dr. Edwards's great contribution to veterinary science and that with which his name always should be associated was the discovery of a method for attenuating the cattle virus, by passage, at first in rabbits and later in goats. It could then safely be inoculated alone, without simultaneous injection of antiserum, in the active immunisation of relatively resistant breeds of cattle indigenous to the plains of India. Some years later a former Director-General of the Indian Medical Service wrote of him and his work at this time : "I have never met a more enthusiastic research worker. Dr. Edwards never spared himself, and his work on the 'goat virus' method of immunisation against rinderpest is now regarded by competent authorities as likely to prove a solution to the vast rinderpest problem of India." However, Edwards's work on rabbit passage of the virus was not to remain unfruitful, Nakamura in Japan developing what is now known as the lapinized strain of rinderpest virus, the use of which is increasing rapidly to-day. Moreover, Edwards's work on rinderpest has assisted in recent studies of the swine fever virus. Within the last six years success has attended experiments to adapt the swine fever virus to rabbits, and strains of the rabbit-adapted virus are now being developed as a greatly improved method of immunisation against swine fever. Other research work undertaken included bovine haemorrhagic septicaemia, blackquarter, anthrax and piroplasmoses (bringing the identification of Theileria for the first time in India) ; also the study of the rôle of these infections in the acclimatisation of imported cattle—a vital matter where any attempt to grade up the native breeds is concerned—while equine strangles, glanders and surra did not escape his devouring appetite, and aptitude, for study.

It was during the second year of his stay in India that Dr. Edwards married the second daughter of Sir John M'Fadyean. She went out to him and the wedding took place in Bombay Cathedral, with Grahame Williamson as best man.

Returning from India in 1929 Dr. Edwards began work for the Foot-and-Mouth Disease Research Committee of the Ministry of Agriculture at the Lister Institute, Chelsea. After four years there he joined the staff of the Research Station for Foot-and-Mouth Disease at Pirbright, in August, 1933. At both centres he was found to be, as we well can believe, "an indefatigable research worker," and it is also said "one who has added considerably to our knowledge of foot-and-mouth disease by his intensive and persistent work with all available means."

Egypt

Another great, and again a literal, departure followed, involving leave of absence from the Ministry. In January, 1946, Dr. Edwards was invited by the Egyptian Government to visit the country as Veterinary Expert to enquire into and report upon the Veterinary School at Giza on behalf of Fuad I University. At the termination of this mission he was invited by the University to remain to help implement the principles and proposals enunciated in his report, occupying for that purpose the temporary posts, first of Dean of the newly instituted Faculty of Veterinary Science and then of Visiting Professor at the School. During the earlier period of his stay he also rendered assistance, on request, to the Egyptian Minister of Agriculture in combating cattle plague (rinderpest) which was then spreading in the country. Measures were being worked out for control of the disease when Dr. Edwards, his main mission completed, returned to England in March, 1947. Tributes from the University Council were supplemented by the Minister of Agriculture's expression of gratitude "for all the good work you have done for the School and for the Veterinary Services of Egypt," while in July, 1948, His Majesty King Farouk conferred upon him the Order of the Nile (Third Class) "in recognition of valuable services rendered to Egyptian interests in the field of veterinary science."

Literary Work

Mention already has been made of Dr. Edwards's supremely important services to this journal from his early years to the time of his last illness, not least as a member of the Editorial Committee. The background of this loyal endeavour is well worth filling in. He was present at the meeting in

1919 when the decision was taken to launch the Association in its present form, whereby British Veterinary Societies at home and abroad were linked up in a parent Association. Dr. Edwards at once placed his literary and journalistic abilities at the service of the Association's journal, which it had acquired in 1913 on the death of William Hunting, the founder, for there was an essential post to be filled on the death of the Secretary-Editor, John Malcolm, in 1920. Despite his pressing commitments as a member of the staff of the Royal Veterinary College, Dr. Edwards, as Honorary Editor, managed not only to fulfil the supremely difficult task of bringing out a weekly periodical still printed in Birmingham, but to enhance its reputation.

When, in 1921, Dr. Edwards left for an eight-year period of service in India, he left on a secure footing a journal now printed in London, under a strong Editorial Committee, with a new Editor and the present incumbent continuing as Assistant Editor. He rejoined the Committee in 1933, serving continuously since except for his sojourn in Egypt from 1946 to 1948.

This latter period has been marked by the publication in the *Record* by Dr. Edwards of several historical studies—involving an immense amount of literary research. Outstanding amongst these was the King George V Silver Jubilee celebration article in 1935, the *Record* Jubilee number in 1938, and the R.C.V.S. Centenary number in December, 1945—a task which took nearly two years to complete. Moreover, in January of last year he contributed to our columns a four-instalment article, "Veterinary Medicine During the Past 50 Years," to mark the close of the half-century. Dr. Edwards was also a valued contributor of historical studies to the *British Veterinary Journal*, and on occasion to continental veterinary periodicals. His literary labours included, not only the joint editorship of the *J.C.P.T.*, but also a period (1916 to 1921) as Editor of the *Tropical Veterinary Bulletin*, an abstract journal published quarterly by the Bureau of Hygiene and Tropical Diseases. He supplied all the matter, surveying the whole field of diseases in animals in the tropics—for which purpose, in addition to summarising English articles, he translated from French, German and Italian journals. He was Correspondant étranger de l'Académie de Médecine de France.

Dr. Edwards's eminence as a veterinary scientist was recognised by the N.V.M.A. in the award to him of the Weldon Dalrymple-Champneys Cup and Medal in 1940, while his voice and influence have been prominent in the Council of the Association since 1941; for long he represented the Central Veterinary Society, which presented him with its "Victory" Medal in 1938. Dr. Edwards has a wide experience as an Examiner, serving in this capacity the Royal College (for the Fellowship Diploma and the D.V.S.M.) and the University of London (for the B.Sc. degree). He was a Fellow of the Royal Society of Tropical Medicine and Hygiene; also a member of the Pathological Society of Great Britain and Ireland and of the Biochemical Society.

This epitome of a life of service fittingly may be concluded with a statement of Dr. Edwards's vision for the profession, as seen by one who served, not only the Council of this Association, whose interests he did so much to promote, but as a member also of the Governing Body of the profession since 1946. It is clear that he thrilled to the *momentousness* of this period in our history. Hear him in his natural vein:

"In this year" (1951) "the Council of the R.C.V.S. has become armed by statute with certain powers which it never had before and which it must sacredly and vigilantly discharge; meanwhile, it has become hedged in, too, in its functions by that self-same powerful statute the Act of 1948. . . . It is the stern duty of all who toil in the service of the profession to acquit themselves creditably in preserving the heritage won for them. . . . But, in the life of the profession, it is the 'National,' above all, which has become the free voice, the unfettered political soul, of the profession. . . . We shall hold together and achieve our goals, so long as we discern what is our common interest."

So he leaves us, with a prophecy worthy of his own faith and vision, a self-sacrificing example for our following, and a call so to equip and conduct ourselves that we may be enabled worthily to meet the challenge of our time.

The deep sympathy of the profession will be extended to the bereaved family. Dr. S. J. Edwards, F.R.C.V.S. (brother), is a well-known member of the staff of the Agricultural Field Station at Compton, in Berkshire, and a member of the Council of our Association.

At its meeting at the Connaught Rooms on Friday of last week the proceedings were interrupted for a two-minute, somewhat stunned, silence of heartfelt tribute, as the President made the sad announcement kindly brought in person from Red Lion Square by the Registrar of the Royal College.

LIVESTOCK EXPORT GROUP

Report for the Quarter Ended September 30th, 1952

This report includes the following information:—

Exports—General.—It was inevitable that with the continuance of foot-and-mouth outbreaks throughout the summer months with attendant embargoes placed on the importation of stock from Great Britain by a number of countries, we should reach a period during the year when no animals would be exported through quarantine, and it is with regret that the Group announces that no animals were so exported during the quarter under review. However, in spite of the situation, only few orders have had to be cancelled—the quarantine register still shows over 600 head of stock for Canada and the U.S.A., over 100 for Australia and some 30 head for New Zealand.

No doubt, now that the foot-and-mouth epidemic has ended—and it is reasonable to suppose that it has—the export of stock will gradually revert to its former even flow and, perhaps, if one can judge by overseas enquiries, particularly from the West, reach a new high level.

Australia.—Members will be heartened to learn that the Group was recently advised by the Australian authorities that, in view of the general abatement of foot-and-mouth disease in Great Britain and the removal of the fear of the spread of infection, they have now considered it safe to remove the embargo placed on the importation from Great Britain of cattle and that, subject to a satisfactory report being received from here, they would be prepared to recommend that the shipment of cattle may be resumed from November 1st, 1952.

South Africa and Southern Rhodesia.—A further happy note is struck by the decision of the Union authorities to lift their ban on the importation of stock—this order is immediately effective. Here, again, a large backlog exists—282 head of cattle, sheep and pigs—which will take some time to reduce. This announcement may come as a surprise as only a short while ago the Union authorities announced, after considering the matter in September, that they had decided to discontinue shipments until after December 31st, when they would review the position again.

Canada and U.S.A.—No indication can yet be given as to when the Canadian authorities are likely to re-open their ports to livestock. It is understood, however, that high-level discussions have been taking place on the matter and as soon as information becomes available an announcement will be made.

For the moment, we can only hope that Canada will follow the lead of other countries who, by their decision to raise embargoes, imposed by them because of the recent prevalence of foot-and-mouth disease, confirm the belief that the epidemic has now ended and the fear of infection from the disease has diminished.

Glasgow Quarantine Station.—The latest stage reached regarding the enlargement of the Glasgow quarantine station is that plans and estimates have been prepared for the work to be carried out and Treasury sanction is now awaited. It is estimated the work will take about two months to complete.

ABSTRACTS

Human Salmonella Infection Due to Duck Eggs*

Two fatal and two severe cases of food poisoning attributed to the eating of duck eggs and the subsequent bacteriological investigations are described. In two cases the incriminated eggs were successfully traced to two flocks of ducks. The probable source was traced in the third case but not in the fourth.

From the first fatal case (a man, aged 58, who had eaten a raw duck egg) *Salm. typhi murium* Vi phage Type 2 was isolated from the stomach, small bowel, faeces and urine. Organisms of the same phage type were isolated from two of nine eggs laid by the incriminated flock of ducks. The flock (seven ducks and one drake) was destroyed and similar organisms were recovered from the ovary of one duck and from both the ovary and intestinal tract of a third duck. The agglutinating titres showed that all the birds except the drake and one of the ducks were positive.

From the second fatal case (a woman, aged 56, who had eaten a lightly poached duck egg) *post-mortem* cultures yielded *Salm. typhi murium* Vi phage Type 4. No specimens were available from the incriminated flock of 24 ducks amongst which there had not been any deaths or illnesses.

The first non-fatal case concerned a woman aged 30 who had eaten a fried duck egg. *Salm. typhi murium* Vi phage Type 4 was isolated from her faeces. Organisms of the same phage type were isolated from one of the 39 ducks of the incriminated flock. The owner of the ducks and his sister, who some time before had been ill after eating ice-cream containing duck eggs, were both found to be excreting *Salm. typhi murium* of the same phage type as the strains isolated from the duck and the patient. An examination of blood samples from the ducks showed that almost the whole flock was infected.

The second non-fatal case concerned a man aged 43 who had eaten a raw duck egg which had passed through a packing station. *Salm. typhi murium* phage Type 4 was isolated. Subsequent investigations into the probable origin of the egg were difficult. All the ducks in two moderate sized flocks (47 and 27 birds respectively) and a proportion (24 birds) of one large flock of 3,000 ducks were examined. Examination of the cloacal swabs and eggs from the flock of 47 ducks was negative but 18 were positive for *Salm. typhi murium* agglutinins. Examination of the flock of 27 ducks was negative. Similarly, the examination of a portion of the large flock was negative for *Salm. typhi murium*, but two of the egg-shells yielded a profuse growth of *Salm. thompson*.

J. S. P.

* **Human Salmonella Infection Due to Duck Eggs.** Miller, A. A. (1952.) *Brit. med. J.* No. 4776. 125-127.

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Mercurochrome as a Semen Stain*

The author tabulates four formulae using mercurochrome as a semen stain for morphological study in routine artificial insemination work.

He concludes that a 1 per cent. aqueous solution shows great promise and is to be highly recommended for its ease of use, reliability, clarity of staining and uniformity of result. Smears of semen were made, fixed by air drying and 1 per cent. aqueous solution mercurochrome was applied for 15 to 30 seconds. No clearing being needed, the smears were washed in running tap-water and dried by blotting or air drying. The resultant stain was excellent, the spermatozoa alone being stained and showing a bright pink against a white background ; it remained without fading for over a year.

J. C. V. H.

* **Mercurochrome as Semen Stain.** Rao, C. Krishna. (1952.) *Ind. Vet. J.* **28.** 423-424.

REVIEW

[Sheep Diseases. By J. E. Newsom. Pp. x + 352, with 118 figures. Published by Baillière, Tindall & Cox, Ltd., London. 1952. Price 54s.]

The value of this book, written by an American author, is difficult to assess, particularly with knowledge based on British conditions, but it is comprehensive and most of the common and some of the less common diseases of sheep are included. The arrangement of the subject matter is on conventional lines, namely, diseases due to bacteria, fungi, protozoa, filterable viruses, rickettsiae, external parasites, internal parasites, deficiency diseases, diseases of the various anatomical systems and poisonings.

Although many diseases are described which do not occur in the U.S.A., as is to be expected, the main emphasis is on American conditions. Thus in the section on ecto-parasitic infestations there is a discussion on tick paralysis as it occurs in the U.S.A., where it is associated with the tick *Dermacentor andersoni*, but no mention is made of the importance of *Ixodes ricinus* as a disease vector, although it is mentioned briefly under louping-ill.

The intoxications due to the clostridia are well described, but again there appear to be differences in outlook which may be confusing to the British reader. In the treatment of lamb dysentery consideration is given to sulphathalidine, sulphaguanidine and acriflavine. The use of vaccine in the ewes or antiserum in the lambs is recorded under preventive measures, but the section concludes, " In this country, then, the control of lamb dysentery seems to be a sanitation problem," and analogies are drawn with scour in calves. This hardly coincides with British experience. A general discussion of the antigenic structure of the *Cl. welchii* types and their laboratory diagnosis would be helpful in this section for a better understanding of the enterotoxaemias.

The description of Johne's disease in sheep does not mention the characteristic yellow appearance of the intestinal mucosa which has been described in one form of this infection, and one would also hardly agree with the statement that " giant cells, while frequently present in cattle lesions, . . ."

These are minor criticisms of a book which shows a wide knowledge of disease in sheep coupled with an extensive acquaintance of the literature, including recent papers. In this latter connection it is refreshing to read an American book which gives adequate acknowledgment in its references to work done in other countries. The references include many names well known in this country and Australasia. The book is well produced and the photographs are excellent. " Sheep Diseases " will be appreciated by many of those interested in sheep and it will be of value to those able to allow for the differences in approach and experience resulting from work in another country.

QUESTIONS IN PARLIAMENT

Slaughterhouses (Siting Policy)

Mr. SPIER (November 5th) asked the Minister of Food whether he is yet in a position to make a statement about the Government's policy on the siting of slaughterhouses.

Major LLOYD GEORGE: Yes. It is essential that the construction of new slaughterhouses and the modernising of old ones should proceed as quickly as our resources permit and I have therefore agreed with my agricultural colleagues that, while we cannot yet make any announcement about the important subject of livestock marketing policy, we should indicate the lines on which we propose that the future siting of slaughterhouses should develop.

The Government have no intention of continuing indefinitely the present arrangements under which the Ministry of Food controls and operates the slaughterhouses used for preparing our home-produced meat. That must go on while meat rationing is necessary; but it is our intention that local authorities and private interests should take over the responsibilities as soon as circumstances permit. It is essential, however, that there should be some central regulation to ensure that buildings in which slaughtering is carried on meet all the requirements of hygiene, meat inspection and humane slaughter and that they should be so sited as to make it possible to achieve these objectives with reasonable economy, and also to ensure the maximum economies in the total cost of transport of both livestock and meat.

A return to the pre-war system of slaughtering in thousands of small premises would not do that, nor would the building of large factory-type plants be suitable for the needs of this country. The Government therefore propose in this respect to continue the policy announced by the previous Government of what is generally known as moderate concentration, that is, concentration into some 300 or 400 slaughterhouses throughout Great Britain. The exact number cannot, of course, be determined at once.

In order to work towards a system of this sort, for which amending legislation may ultimately be necessary, my Department will provide the necessary machinery, in consultation with other Departments and interested organisations, for drawing up a national siting plan. We shall call into early discussions representatives of local authorities, producers, butchers and others, and we shall invite the co-operation of professional bodies concerned with these matters. The arrangements in Scotland will be similar to those in England and Wales and will be made by my right hon. Friend the Secretary of State in consultation with me.

NOTES AND NEWS

Diary of Events

Nov. 15th.—Meeting of the Biochemical Society, in the London School of Hygiene and Tropical Medicine, Keppel Street, W.C.1, 10.30 a.m.

Nov. 20th and 21st.—B.V.A. Conference on Metabolic Disorders and Other Problems Related to Grassland and Fodder Crops and Innovations in Animal Husbandry to be held in the Auditorium of the Wellcome Foundation, 183, Euston Road, London.

Nov. 20th.—Research Defence Society's 21st Stephen Paget Memorial Lecture, in the Physiology Lecture Theatre, University College, Gower Street, W.1 (Sir Howard Florey, M.D., F.R.S., "The Advance of Chemotherapy by Animal Experiment"), 5.30 p.m.

Nov. 20th.—Meeting of the Herts & Beds Division, B.V.A., at Luton (The George Hotel), 2.30 p.m.

Nov. 26th.—Annual General Meeting of the North Wales Division, B.V.A., at Ruthin (Council Chamber), 2 p.m.

Nov. 28th.—Meeting of the Royal Counties Division, B.V.A., at Reading (Caversham Bridge Hotel), 2.30 p.m.

Dec. 9th.—Technical Development Committee, B.V.A. (to discuss Sheep Diseases Report), 11 a.m.

Dec. 10th.—Technical Development Committee, B.V.A. (Ordinary Meeting), 10 a.m.

Dec. 12th.—Annual Dinner and Dance, North Wales Division, B.V.A., at Llandudno. (See Notice.)

Jan. 9th, 1953.—Dumfries and Galloway Division, B.V.A., Ladies' Guild: Annual Dance in aid of the V.V.B.F., at Dumfries (Cairndale Hotel). (See Notice.)

R.C.V.S. Examinations

Membership Examinations.—Written: Thursday and Friday, December 4th and 5th, 1952. Orals and Practicals: Commencing Monday, December 8th, 1952.

Anthrax

Readers of *The Veterinary Record* will be aware that there has been an increase in the number of outbreaks of anthrax in recent months. The number of animals affected, however, is not on a scale that would justify a widespread campaign of vaccination. But in view of the fact that the outbreaks—particularly among pigs—have reached a higher level than for some years, the Ministry of Agriculture is considering making available for purchase supplies of vaccine to veterinary surgeons who may feel that its use would be justified in certain cases. It will be recalled that it has long been the policy of the Ministry to make such vaccine available through divisional veterinary officers in certain areas such as the Nene Valley of Northamptonshire, where anthrax is endemic. It is understood that the new vaccine is more efficacious than that previously in use and, moreover, is harmless.

A glance at the figures below shows the extent to which the number of pigs affected has increased the scope of the present outbreak. While it cannot be scientifically proved, it seems likely that the increase in the use of balanced rations for pigs, involving, as it does, a proportion of imported feeding-stuffs, is the basic cause of the increase in anthrax among these animals.

It would appear that, short of the total prohibition of imported feeding-stuffs, which seems to be an economic impossibility at the present time, there is no complete answer to the problem. The virtual certainty that such feeding-stuffs are the main source of infection is clearly illustrated in the statistics, where it will be noticed that, during the war years, when imports fell to a minimum, the number of outbreaks fell correspondingly. They began to rise again as soon as imports recommenced.

Practitioners may find it useful to recall to their farmer clients the essential precautions that must be taken when an animal dies of anthrax. These include great care not to spill blood, not to attempt salvage of the carcase and hide, and not to dispose of the carcase to a knacker unless the cause of death is quite definitely not anthrax—all, it should be emphasised, illegal practices. If, as sometimes happens, the carcases of animals which have died from this disease are opened in ignorance, it may be sound practice to have all stock on the affected farm vaccinated.

ANTHRAX OUTBREAKS

	1949	1950	1951	To Oct. 27th, 1952
Number of outbreaks ..	244	344	407	815

ANIMALS ATTACKED

	1949	1950	1951	To Oct. 27th, 1952
Cattle	247	338	410	532
Sheep	1	3	—	1
Swine	12	20	21	373
Horses	2	6	1	2
Felines	2	—	1	—
Other animals	—	605 Mink	7	4
Dogs	—	—	—	2

NUMBER OF OUTBREAKS MONTHLY

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	To Oct. 27th, 1952
1952	42	32	45	55	69	31	90	123	170	158
1951	56	49	41	48	42	24	15	24	20	—

NUMBER OF OUTBREAKS INVOLVING SWINE

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	To Oct. 27th, 1952
1952	2	—	1	7	11	6	56	64	97	75

PROBABLE ORIGIN OF OUTBREAKS JAN. TO OCT. 27TH, 1952

Number of outbreaks	815
Artificial feeding-stuffs ..	662
„ fertilisers	31
Combination of above	32
Obscure	34
Previous outbreaks	14
Tanneries, etc.	17
Incomplete	25

Anthrax

Total Number of Confirmed Outbreaks each Year from 1931 to October 13th, 1952 (inclusive), and Number of Animals attacked during the Years 1938 to October 13th, 1952 (inclusive)

Year	1931	1932	1933	1934	1935	1936	1937	1938	1939	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949	1950	1951	Oct. 27th 1952	Total
Outbreaks	465	344	297	395	386	468	743	830	699	564	399	372	283	189	119	95	121	118	244	344	407	815	8,697
Cattle ..	—	—	—	—	—	—	—	855	735	620	436	404	311	198	131	96	127	117	247	338	410	532	5,557
Sheep ..	—	—	—	—	—	—	—	3	2	3	1	1	—	1	—	—	—	—	1	3	—	1	16
Pigs ..	—	—	—	—	—	—	—	45	45	41	11	3	5	2	1	—	5	11	12	20	21	373	595
Horses ..	—	—	—	—	—	—	—	8	3	6	3	6	2	2	1	1	5	—	2	6	1	1	47
Canines ..	—	—	—	—	—	—	—	2	1	—	—	—	—	1	2	1	2	—	—	—	—	3	12
*Felines	—	—	—	—	—	—	—	—	—	6	9	—	—	—	—	—	—	—	2	—	1	—	18
Mink ..	—	—	—	—	—	—	—	315	—	—	—	—	—	—	—	—	—	—	—	605	—	—	920
Ferrets ..	—	—	—	—	—	—	—	7	6	—	—	—	—	—	—	—	—	—	—	—	—	—	13
*Other animals	—	—	—	—	—	—	—	—	0	1	10	—	2	—	—	—	—	—	—	—	7	1	21
Total No. of animals	—	—	—	—	—	—	—	1,235	792	677	470	414	320	204	135	98	139	128	264	972	440	911	7,199

* Mainly Zoological species.

Allocation of New Cars

The B.V.A. has on its files the names of many members whom it has tried to assist in obtaining new cars but from whom it has had no communication for some time. The General Secretary would like to hear from these members whether they have now taken delivery of the car for which a priority certificate was issued or whether they require any further help.

* * * * *

Control of Distribution of Biological Products

The profession will know from reports made to Council, B.V.A., of the discussions which have taken place over a period to obtain some measure of general agreement on the control of distribution of certain biological products for veterinary use. The outcome of these discussions was that the Pharmaceutical Society agreed to put a level of control into operation through its members and it has now issued the following statement:—

Pharmaceutical Society of Great Britain
Restrictions on Supply of Products for Veterinary Use

Statement by the Council

Among various matters which have been discussed recently by representatives of the Society and the veterinary profession has been control over the production and supply of veterinary medicines. At the present time no legislation comparable with that applied to human medicine is in existence and the situation is being studied with a view to proposals for appropriate legislation being made. Pending such legislation being enacted consideration has been given to the extent to which pharmacists could assist in bringing about an improvement in the situation. It is recognised that the activities most in need of attention take place outside pharmaceutical control but it is considered desirable that in so far as pharmacists can influence the situation for the better they should act accordingly.

In considering what might be done in this direction attention has been given to the growing range of biological and chemotherapeutic products which should be administered only by or under the directions of a veterinary surgeon if the danger of their being misapplied to the detriment of the animals concerned is to be avoided. The appended list of products of this nature has been drawn up in agreement with the veterinary profession and the Council have decided to ask pharmacists not to supply such products for animal treatment or associate themselves directly or indirectly with such supply except to or on the prescription of a veterinary surgeon or veterinary practitioner. By so doing they will ensure that such influence as pharmacists can bring to bear upon the situation at this stage will have the effect of raising the standard of animal care.

The Council furthermore are satisfied that the matter should be regarded as one of professional conduct and if, after due warning has been given, information reaches the Council of any pharmacist acting in contravention of the request, they may feel it necessary to take disciplinary action.

List referred to:—

Anthrax vaccine and serum.
Anti-piroplasmosis agents.
Autogenous vaccines.
B. coli (white scour) vaccine and serum.
Bronchisepticus antiserum.
Brucella abortus vaccine.
Canine distemper prophylactics and serum.
C. pyogenes toxoid, vaccine and serum.
Haemorrhagic septicaemia vaccine and serum.
All hormone products.
Iodinated proteins.
Joint-ill vaccine.
Leptospira vaccine and serum.
Mallein.
Mastitis vaccine.
Pneumonia and strangles vaccines and sera.
Staphylococcus toxoid.
Swine erysipelas vaccine and serum.
Swine fever (crystal violet) vaccine.
Swine fever serum.
Synthetic oestrogens other than preparations used for the chemical caponisation of poultry.
Tetanus toxoid and serum.
Thiouracil and related compounds.
Tuberculin.

[The inclusion of a product in this list is not intended to imply any recommendation for its use in prophylaxis or treatment.]

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PERSONAL

Mr. Albert Messervy's Appointment to Bristol Chair of Veterinary Surgery

Announcement is made that Mr. Albert Messervy, M.R.C.V.S., has been appointed to the new Chair of Veterinary Surgery in the University of Bristol.

A London (1929) graduate, from that year until 1941 Mr. Messervy was in general practice. In 1941 he was appointed Senior Lecturer in Veterinary Surgery and Obstetrics at the Royal Veterinary College a post which he held until 1945. During the last three years of this period he acted as the veterinary surgeon in charge of the Royal Veterinary College practice at Wallingford, Berkshire. In 1945 he returned to Jersey as the senior member of a mixed general practice. He has published a number of articles on veterinary subjects and has acted as external examiner in surgery and obstetrics to the Royal College of Veterinary Surgeons and to the University of Liverpool for the B.V.Sc. degree. In 1951 he was elected President of the World Jersey Cattle Bureau, being re-elected in August of this year.

Mr. Messervy will take up the post on January 1st.

Veterinarian's Model Engineering Award.—Further proof that Mr. J. W. Burgess, M.R.C.V.S., Lecturer in Surgery at the Royal (Dick) School of Veterinary Studies, Edinburgh, is one of the most successful model engineers in the country, was forthcoming at the Model Engineers Exhibition recently held in the Agricultural Hall, Islington, N. Entering a universal dividing head, Mr. Burgess was awarded a diploma in the machine tool and workshop appliances section.

Will.—Mr. William Frederick Maynard, of Crescent Road, Romsey, Hants, veterinary surgeon (£9,031 gross), £8,974.

Births.—Bell.—To Edith, wife of R. L. Bell, Briarlea, Longtown, a son.

Gourley.—On October 16th, 1952, to Ruth, wife of Jim Gourley, B.Sc., M.R.C.V.S., twins, Michael and Sheila—brother and sister for Ian.

Owen.—On November 1st, 1952, at Bryn Beryl Hospital, Pwllheli, to Lona (*née* Lewis), wife of Desmond C. Owen, M.C., M.R.C.V.S., a daughter—Cathrin Ann.

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R.C.V.S. OBITUARY

Edwards, James Thomas, D.Sc. (Lond.), M.R.C.V.S., Captain late R.A.V.C. (S.R.), Correspondant étranger de l'Académie de Médecine de France; Order of the Nile (3rd class); of 23, Alwyne Road, Canonbury, N.1. Graduated London, July 20th, 1911. Died on Thursday, November 6th, 1952; aged 63 years.

Death of Dr. J. T. Edwards, D.Sc., M.R.C.V.S.

We record above, with profound regret, the death of Dr. James Thomas Edwards, which occurred suddenly as he was travelling home by 'bus from a meeting of the Central Veterinary Society. As our readers will be aware, Dr. Edwards was seriously ill in the late summer, but he had been discharged from University College Hospital for some weeks when a weakened heart took its toll. A memoir of this distinguished veterinary teacher and scientist, who was our first editor under Association ownership of this journal, is published elsewhere in this issue. Personal tributes will follow, together with further reference to the funeral, which took place at Golders Green Crematorium on Tuesday last.

The Late Mr. Louis G. Gryspeerdt, M.R.C.V.S.

Mr. Louis George Gryspeerdt, M.R.C.V.S., of Croham Park Avenue, Croydon, whose death at the aged of 72 years we announced with much regret last week, was educated at St. Edmund's College, Ware, and Melle College, Belgium. After qualifying at the Royal Veterinary College, Camden Town, Mr. Gryspeerdt entered the practice of his father, the late Mr. E. Gryspeerdt.

He served in the first world war as a Captain in the Army Veterinary Corps and took charge of a hospital at Luton.

A keen gardener, he took many prizes for carnations. This year alone he gained 12 first prizes.

He was a member of Croydon Rotary Club and attended St. Gertrude's Church, South Croydon.

Funeral of Mr. David Penman

Mr. David Cameron Penman, whose death we recorded with deep regret in our issue of last week, and who resided at 13, Queen's Crescent, Glasgow, was only 36 years of age. A member of Lenzie Badminton Club, he had just finished a game when he collapsed and died.

The following members of the profession were present at the funeral at the Crematorium, Glasgow, on Saturday, November 1st: Professors J. W. Emslie and W. L. Weipers, Miss Doris Crawford, Messrs. J. T. Cairns, R. S. F. Campbell, W. Gowans, R. McGrath, S. Michna, R. F. Ross and Alex Thomson.

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VICTORIA VETERINARY BENEVOLENT FUND

"In Memory of Mr. F. H. Stainton, F.R.C.V.S."

The Secretary of the Fund gratefully acknowledges the receipt of donations, in lieu of flowers, from:—

Mr. and Mrs. Beaufort-Palmer, Mrs. Brown, Lady Carter, Miss Charlesworth, Miss M. M. Chatterton, Joan E. Clarke, Mrs. Corneford, Croydon Canine Society, Mrs. and Miss Farrar, Dr. M. Franklin, Miss Gibson, Mrs. Halse, Miss Harrop, Kensington Canine Society, Miss La Fontaine, Mrs. Marquand, Mrs. Mason, Mr. and Mrs. Mayo, Miss Metcalfe, Metropolitan and Essex Canine Society, Miss Milton, Miss Redfern, Mrs. Selby-Lowndes, Mrs. Trembath and Mr. R. A. Willett. Total received (*November 6th*, 1952), £28 3s. 0d.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax:

Beds.—Leedon Farm, Leedon, Leighton Buzzard (Oct. 31st).

Berks.—Manor Farm, Lyford, Wantage (Nov. 3rd).

Cornwall.—Trengwainton House Farm, Heamoor, Penzance (Oct. 31st).

Denbighs.—Moorhead Lodge, Holt, Wrexham (Nov. 3rd).

Derbys.—Marsh House Farm, Longnor, Buxton (Nov. 5th).

Essex.—Stanford Rivers Hall, Stanford River, Ongar (Nov. 4th); Theydon Grove, Epping (Nov. 5th).

Glos.—Ampney Park, Ampney-Crucis, Cirencester; Cranhill, Frampton Mansell, Stroud (Nov. 5th).

Hants.—"Newlands Prospect Farm," Monston, Andover; Steyne Farm, Binstead, Alton (Nov. 3rd).

Isle of Wight.—Mersley Farm, Newchurch (Nov. 3rd).

Kent.—Yew Tree Farm, Boughton Aluph, Ashford (Nov. 1st); Stone Castle Farm, Fire Oak Green, Paddock Wood (Nov. 4th); Weald View Farm, Colt Hill, Five Oak Green, Tonbridge (Nov. 5th).

Kirkcudbright.—Meikle Kirkland Farm, Urr, Kirkcudbright (Nov. 5th).

Lancs.—Park Gate Farm, Chipping, Preston (Nov. 11th).

Lincs.—Asholme, Briant Road, Lincoln (Nov. 1st).

Norfolk.—Poplars Farm, Autingham, North Walsham (Nov. 1st); Station Road, Alburgh, Harleston (Nov. 3rd); Pear Tree Farm, Stradbroke, Diss (Nov. 4th).

Salop.—Betton Hall Farm, Ridgewardine, Market Drayton (Nov. 1st).

Somerset.—Clapton Farm, Clapton-in-Gordano, Bristol (Nov. 3rd); Bagborough Farm, Evercreech, Bath (Nov. 4th); Ashill House, Ashill, Ilminster (Nov. 5th).

Suffolk.—Church Farm, Theberton (Nov. 1st); Low Farm, Framsden, Stowmarket (Nov. 4th).

Surrey.—The Wellcome Institute, Beckenham (Nov. 1st); Chart Gardens, Limpsfield, Oxted (Nov. 3rd).

Sussex.—Rowden Pig Farm, Frant, Tunbridge Wells (Oct. 31st); Shawhurst Farm, Coleman's Hatch, Tunbridge Wells (Nov. 3rd); Harwood Farm, East Grinstead (Nov. 4th); Dairy Farm, The Haven, Crawley Down (Nov. 5th).

Wilts.—Mount Sorrell, Broad Chalke, Salisbury (Nov. 3rd); Round Robin Farm, Highworth, Swindon (Nov. 4th); Bonham Farm, Stourton, Warminster (Nov. 5th).

Yorks.—The Homestead, Clint, Ripley, Harrogate; Swindon Manor Farm, Hellified, Skipton (Nov. 1st); Mollin Farm, High Birstwith, Harrogate (Nov. 3rd); Walker Farm, Darley, Harrogate (Nov. 4th).

Foot-and-Mouth Disease:

Sussex.—Willingdon Grove, Langney, Eastbourne (Nov. 4th).

Swine Fever:

Ches.—"The Pines," Dodleston, Chester (Nov. 5th).

Clackmannan.—Arns Farm, Clackmannan, Scotland (Nov. 8th).

Cumberland.—Newbiggin Hall, Culgaith, Penrith (Nov. 1st).

Essex.—Chelmoe House, Great Maplestead, Halstead (Nov. 3rd).

Middlesex.—Cooks Hollow, Enfield; The Bungalow, Springwell Lane, Harefield, Uxbridge (Nov. 3rd).

Norfolk.—West Hall Farm, Middleton, King's Lynn (Nov. 5th); Chapel Lane, Ringstead, King's Lynn (Nov. 6th).

Stirlings.—Easter Bowhouse, Maddiston, Falkirk (Nov 6th).

Fowl Pest:

Cambs.—22, Wimblington Road, Doddington March (Nov. 1st).

London.—10, Idmiston Road, Stratford, E.15; 345, Prince Regent Lane, E.15 (Nov. 4th).

Sussex.—Garsten Farm, Bolney, Haywards Heath (Oct. 31st); Neaves Farm, Hartfield, Tunbridge Wells (Nov. 6th).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

VICTORIA VETERINARY BENEVOLENT FUND

Christmas Appeal

Sir,—At this time of the year it is customary to remind members through your journal of the Christmas Gift Account through which gifts in money and in kind are sent out just before Christmas to the recipients of the V.V.B. Fund.

May we therefore appeal to all members of the profession to send a donation, however small, so that we can again help our recipients to obtain those little extra comforts which mean so much to them at this time of the year. Please make your remittance payable to the V.V.B.F. Christmas Gift Account, and send it to the Secretary, 9, Red Lion Square, London, W.C.1, who will gratefully acknowledge receipt.—Yours faithfully, J. B. Buxton, *President;* H. W. Dawes, *Treasurer;* J. J. Dunlop, *Hon. Secretary*, 10, Red Lion Square, London, W.C.1. *November 11th*, 1952.